The global burden of occupational and environmental respiratory diseases in a changing climate

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2021 EAACI Clemens Von Piquert Award
2025 Alice Hamilton Award

Disclosures

International

- ERS: Ethics and Integrity Committee (Member)
- EAACI: - ExCom (Member)
  - Public, Patients & Outreach Hub (Editor in Chief)
- EU DARWIN Advisory Board (Member)

National

- IRD: Ethics Committee (Member)
- MEDD: Comité prévention et protection (CPP) (Member)
- SFA: Scientific Committee (Member)
- RNSA: Scientific Committee (Member)
- Météo France: Commission Santé (Member)
- Société Pneumologie Langue Française: GT PAPPEI (Member)
- APPA: Scientific Council & Board of Directors (Member)

Section Editor for Environmental Health of ERJ and IJTLD

I'M GOING TO TALK ABOUT

- CLIMATE CHANGE AND WHY AND HOW IT IMPACTS HEALTH, RESPIRATORY HEALTH IN PARTICULAR
- CLIMATE CHANGE RELATED RESPIRATORY OCCUPATIONAL AND ENVIRONMENTAL HEALTH BURDEN
- THE IPF CASE STUDY AS AN EMBLEMATIC MODEL
- RESPIRATORY HEALTH INEQUITIES
- TAKE HOME MESSAGES

Global Burden of Respiratory Diseases (All Causes)

Disease/Condition	Deaths/year	People Affected	Key Drivers
COPD	3.23 million	390+ million	Smoking, air pollution, occupational dust
Asthma	450,000	262 million	Allergens, air pollution, climate
Lung cancer	1.8 million	2.2 million new cases/year	Smoking, asbestos, diesel exhaust
LRI (e.g. pneumonia)	2.5 million	Millions (esp. children & elderly)	Infections, air pollution
Occupational exposure-related	~450,000	Millions (underestimated)	Silica, coal dust, bioaerosols

Global Burden of Disease (IHME, 2020) :

- Respiratory diseases account for **~7.6% of global DALYs** (disability-adjusted life years)

AMERICAN THORACIC SOCIETY DOCUMENTS

The Occupational Burden of Nonmalignant Respiratory Diseases

An Official American Thoracic Society and European Respiratory Society Statement

Paul D. Blanc, Isabella Annesi-Maesano, John R. Balmes, Kristin J. Cummings, David Fishwick, David Miedinger, Nicola Murgia, Rajen N. Naidoo, Carl J. Reynolds, Torben Sigsgaard, Kjell Torén, Denis Vinnikov, and Carrie A. Redlich; on behalf of the American Thoracic Society and European Respiratory Society

This Official Statement was approved by the American Thoracic Society May 2019 and the European Respiratory Society March 2019

Rationale: Workplace inhalational hazards remain common worldwide, even though they are ameliorable. Previous American Thoracic Society documents have assessed the contribution of workplace exposures to asthma and chronic obstructive pulmonary disease on a population level, but not to other chronic respiratory diseases. The goal of this document is to report an in-depth literature review and data synthesis of the occupational contribution to the burden of the major nonmalignant respiratory diseases, including airway diseases; interstitial fibrosis; hypersensitivity pneumonitis;

Results: Workplace exposures contribute substantially to the burden of multiple chronic respiratory diseases, including asthma (PAF, 16%); chronic obstructive pulmonary disease (PAF, 14%); chronic bronchitis (PAF, 13%); idiopathic pulmonary fibrosis (PAF, 26%); hypersensitivity pneumonitis (occupational burden, 19%); other granulomatous diseases, including sarcoidosis (occupational burden, 30%); pulmonary alveolar proteinosis (occupational burden, 29%); tuberculosis (occupational burden, 2.3% in silica-exposed workers and 1% in healthcare workers); and community-acquired pneumonia in working-age adults (PAF, 10%).

Occupational burden of nonmalignant respiratory diseases

Disease	Burden
• asthma	• PAF: 16%
• chronic obstructive pulmonary disease	• PAF: 14%
• chronic bronchitis	• PAF: 13%
• idiopathic pulmonary fibrosis	• PAF: 26%
• hypersensitivity pneumonitis	• occupational burden: 19%
• other granulomatous diseases, including sarcoidosis	• occupational burden: 30%
• pulmonary alveolar proteinosis	• occupational burden: 29%)
• tuberculosis (and 1% in healthcare workers)	• occupational burden: 2.3% in silica-exposed workers
• community-acquired pneumonia in working-age adults	• PAF, 10%

PAF: Population attributable fraction

Occupational burden: attribution in case series, incidence rate ratios (IRRs), or AF within a group

Pando

Diapositive 7

IAM1 Isabella Annesi-Maesano; 16/01/2025

Pando

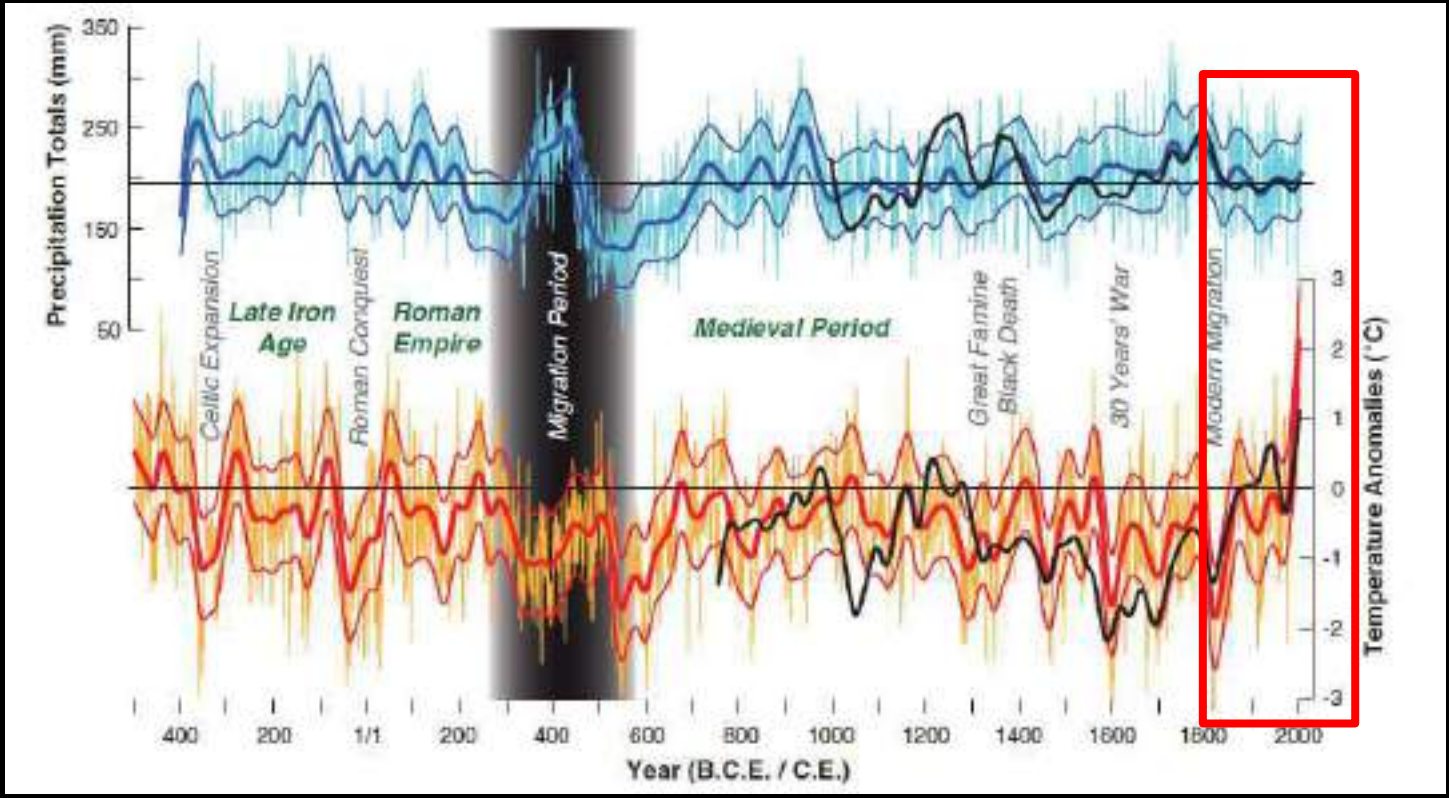
« Pando » = « Je m'étale » = « I spread »

Fishlake National Forest (Utah)

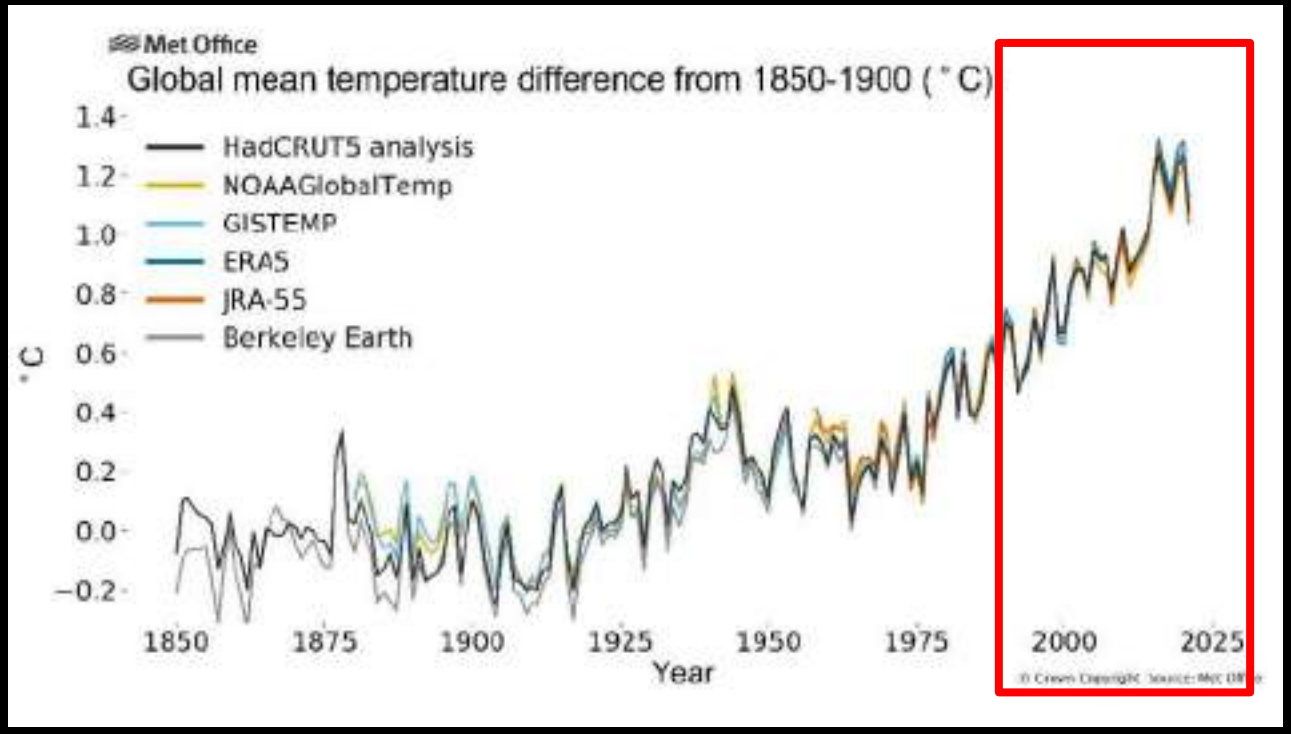
A clone consisting of approximately 47,000 aspen trees (Populus tremuloides) grown from a single seed that spreads by emitting new shoots from an expanding root system. The largest (106 acres), the heaviest, the oldest tree on the planet (>80000 years). One of the most massive living beings on earth.

With 80000 years, I've lived quite a bit

Environmental changes and historical events experienced by Pando

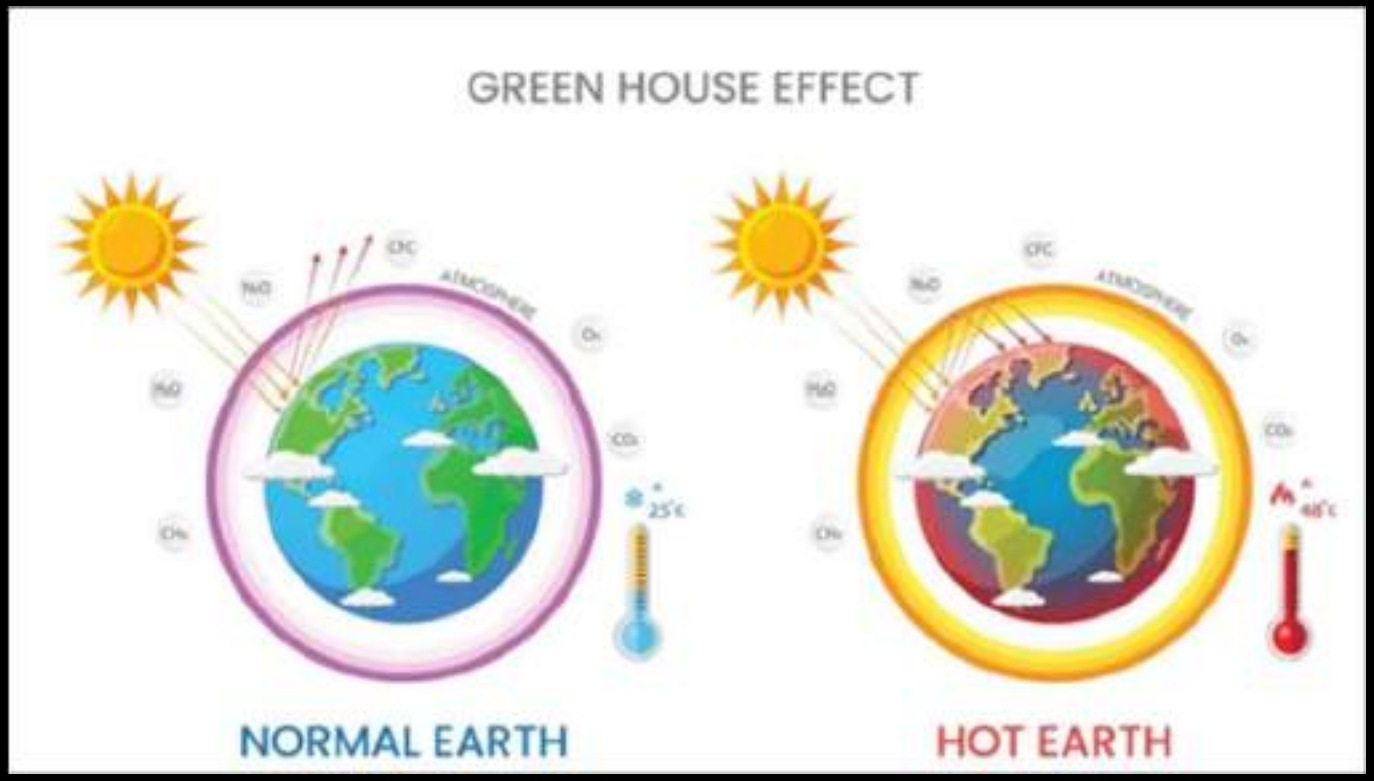


Büntgen U,
Science 2011



Global warming especially in the last 20 years

WHAT HAPPENED?



Greenhouse gases (GHGs):

Carbon dioxide (CO_2)
Methane (CH_4):
Nitrous oxide (N_2O)
Steam
F-gases

"**Normal greenhouse effect**": GHGs trap some of the heat emitted by the Earth after absorbing solar energy essential to maintain a habitable temperature on Earth.

"**Pathological greenhouse effect**": accumulation of GHGs due to human activities

WHO IS RESPONSIBLE FOR WHAT IS HAPPENING?

Those little animals we (the plants) call humans!

Natural and human activities both contribute to the accumulation of greenhouse gases (GHGs)

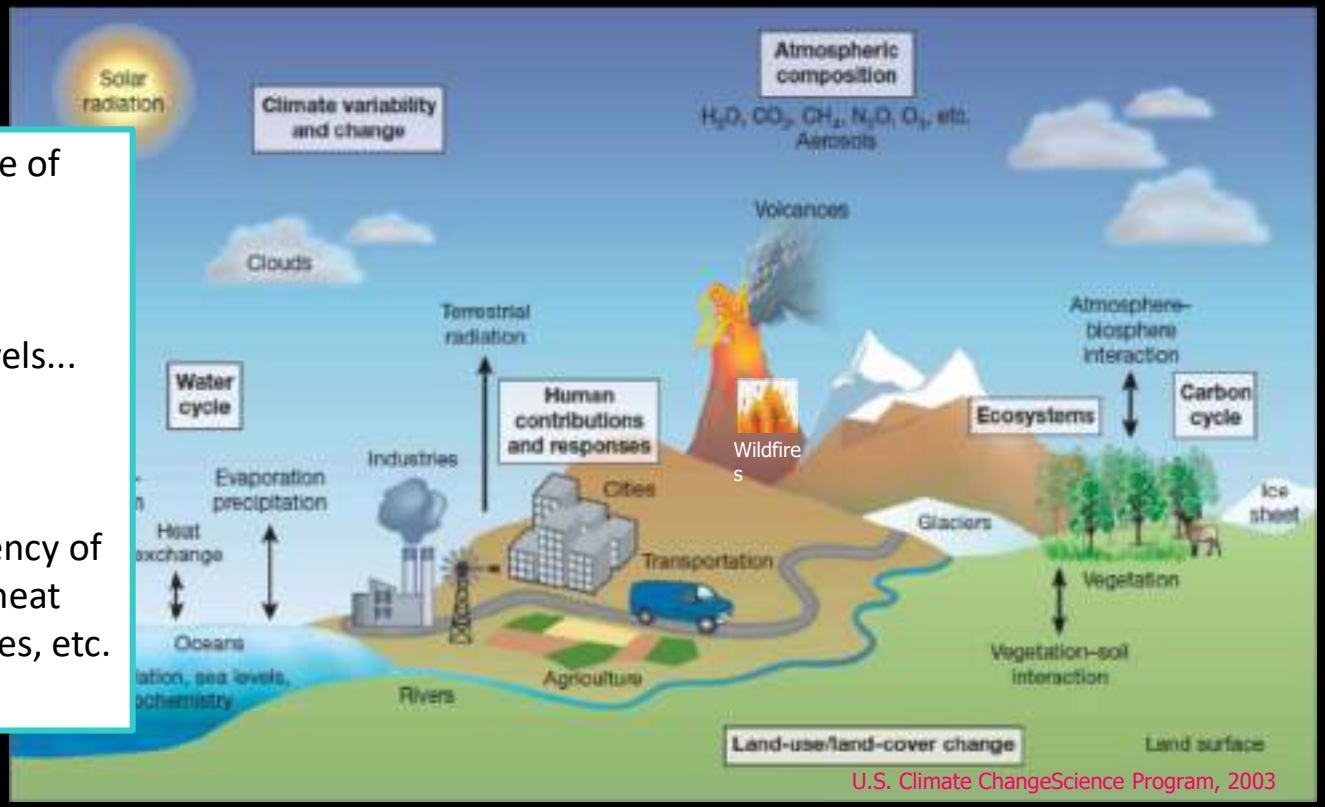
Increase in average temperature of the atmosphere and oceans

↓

Melting snow, ice, rising sea levels...
Modification of the water cycle

↓

Increase in intensity and frequency of extreme climatic phenomena: heat waves, droughts, floods, cyclones, etc.



Even natural phenomena depend on human activities

Acute effects

Incendie à Rome, Juin 2022

Los Angeles, USA, January 2025

Secheresse fleuve Pô, juin 2022

Chronic effects

Il y a 50 ans

Maintenant

Erosion de falaise à Soulac 2023 (200 mt → 20 mt)

WHAT IS THE TREND AND THE CONSEQUENCES OF THE GLOBAL WARMING?

Energy consumption has increased

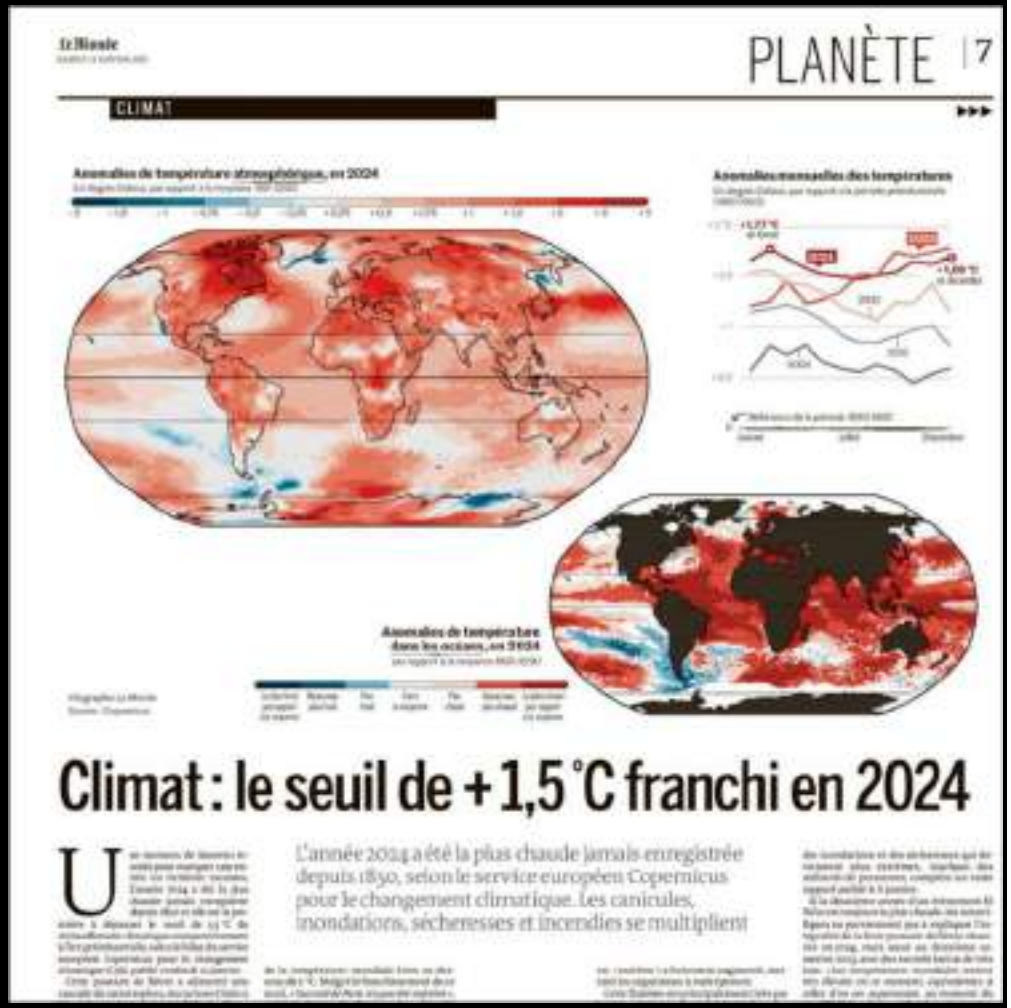
- 17 energy slaves (EGs)* per capita in 1940 (2 billion inhabitants):
- 38 EGs per capita in 1950
- Several hundred EGs to satisfy the consumption of each European (Jancovici and Grandjean) now
- Fossil fuels provide 80% of energy demand

*Tasks once performed by humans or animals are now performed by motors, machines, and automated systems running on energy called energy slaves (R. Buckminster Fuller)

2024

1.62°C exceedance (compared to pre-industrial levels)

N.B.
1.5 °C exceedance => irreversible impacts on ecosystems, human societies and climate.



Le Monde

PLANÈTE 7

CLIMAT

Anomalies de température atmosphérique, en 2024

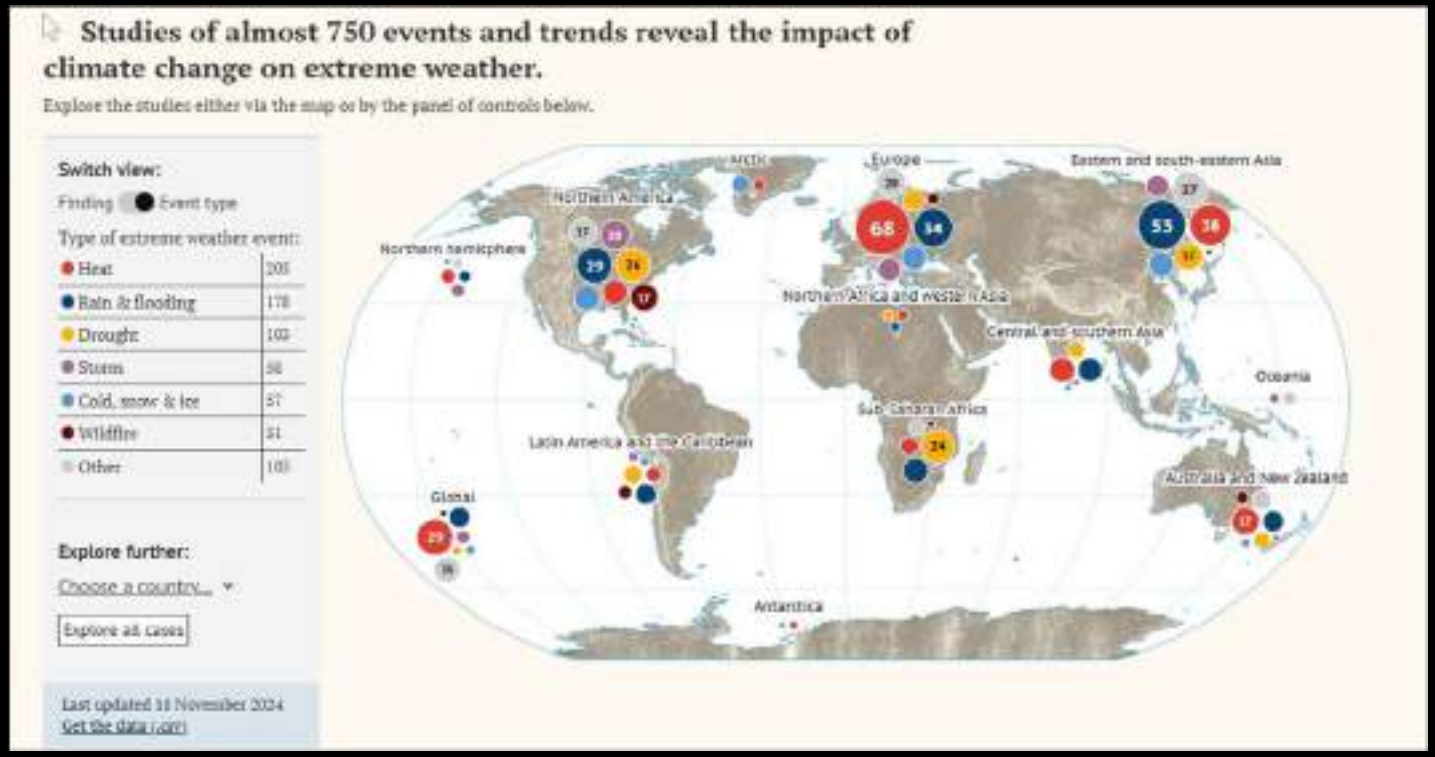
Anomalies mensuelles des températures

Anomalies de température dans les océans, en 2024

Climat : le seuil de +1,5 °C franchi en 2024

L'année 2024 a été la plus chaude jamais enregistrée depuis 1850, selon le service européen Copernicus pour le changement climatique. Les canicules, inondations, sécheresses et incendies se multiplient

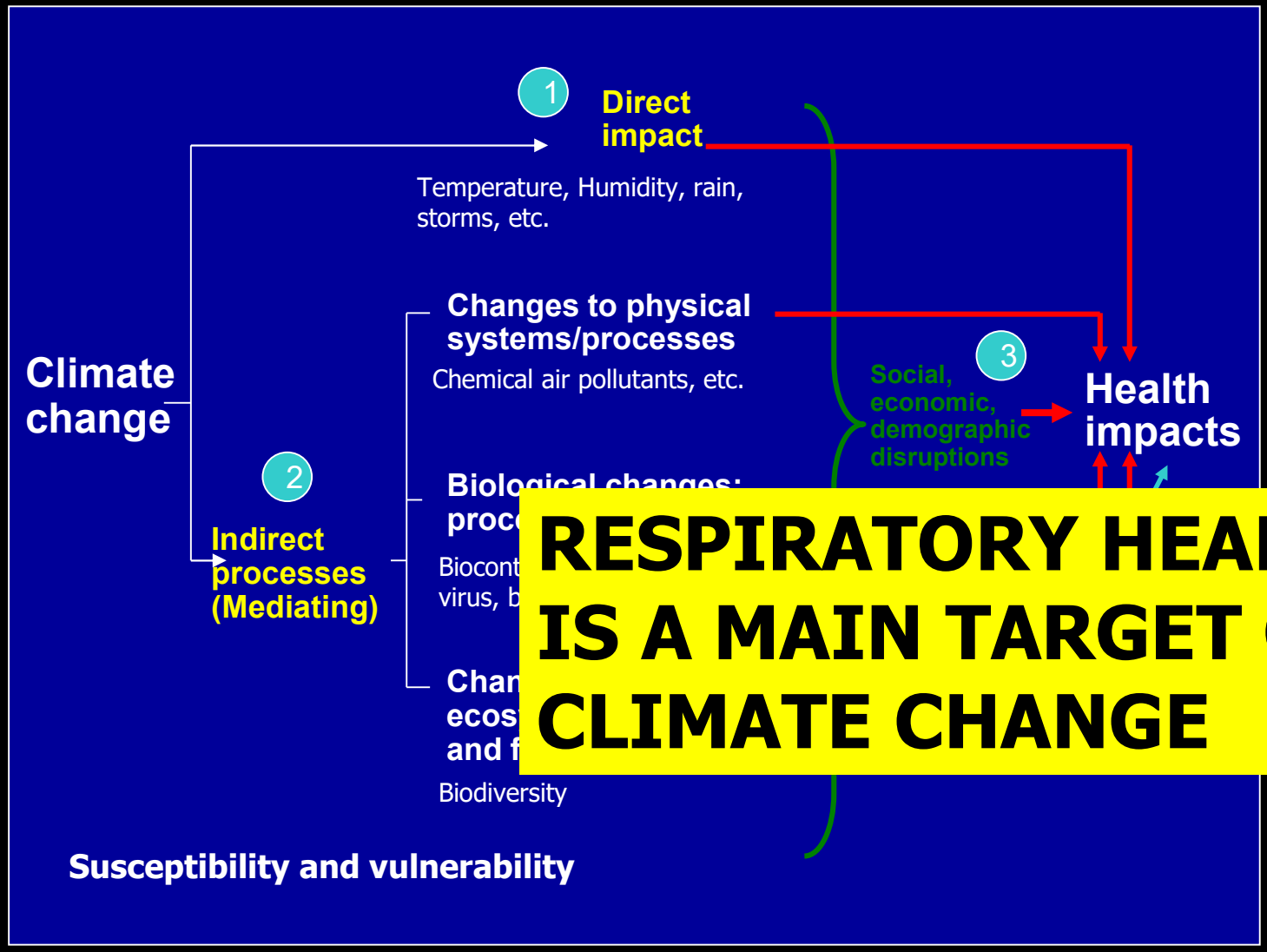
Extreme weather events on the rise



Carbon Brief's map

WHY, THIS IS IMPORTANT FOR HEALTH?

Climate Change-related health pathways



Over 100 million people will die from climate change by 2030 if we do not collectively take action now. Afterwards, the death toll will be even more severe.

Climate change a major driver of both occupational and environmental respiratory diseases

- Reshaping patterns of exposure and disease burden worldwide.

1. Weather patterns impact negatively upon human respiratory health.
2. Worsening air quality and increased allergens and biotoxicants can worsen existing disease or contribute to new disease development.
3. Climate-related changes in vectors for infection can cause new respiratory diseases
4. Increasing occupational pollutants, exposures and conditions contribute to occupational diseases.

THE IMPACT OF CLIMATE CHANGE ON RESPIRATORY DISEASES

Some examples of respiratory diseases impacted by climate change

- Worsening Asthma and COPD:
  - Higher concentrations of O_3 and $PM_{2.5}$ trigger asthma attacks, worsen Chronic Obstructive Pulmonary Disease (COPD) symptoms, and increase hospitalizations.
- Increased Asthma and Rhinitis attacks:
  - Increased pollen and mold exposure trigger asthma and rhinitis
  - Thunderstorm asthma
- Increased Respiratory Infections:
  - $PM_{2.5}$ exposure is linked to increased susceptibility to respiratory infections (e.g., pneumonia, bronchitis).

Some figures of respiratory diseases impacted by climate change

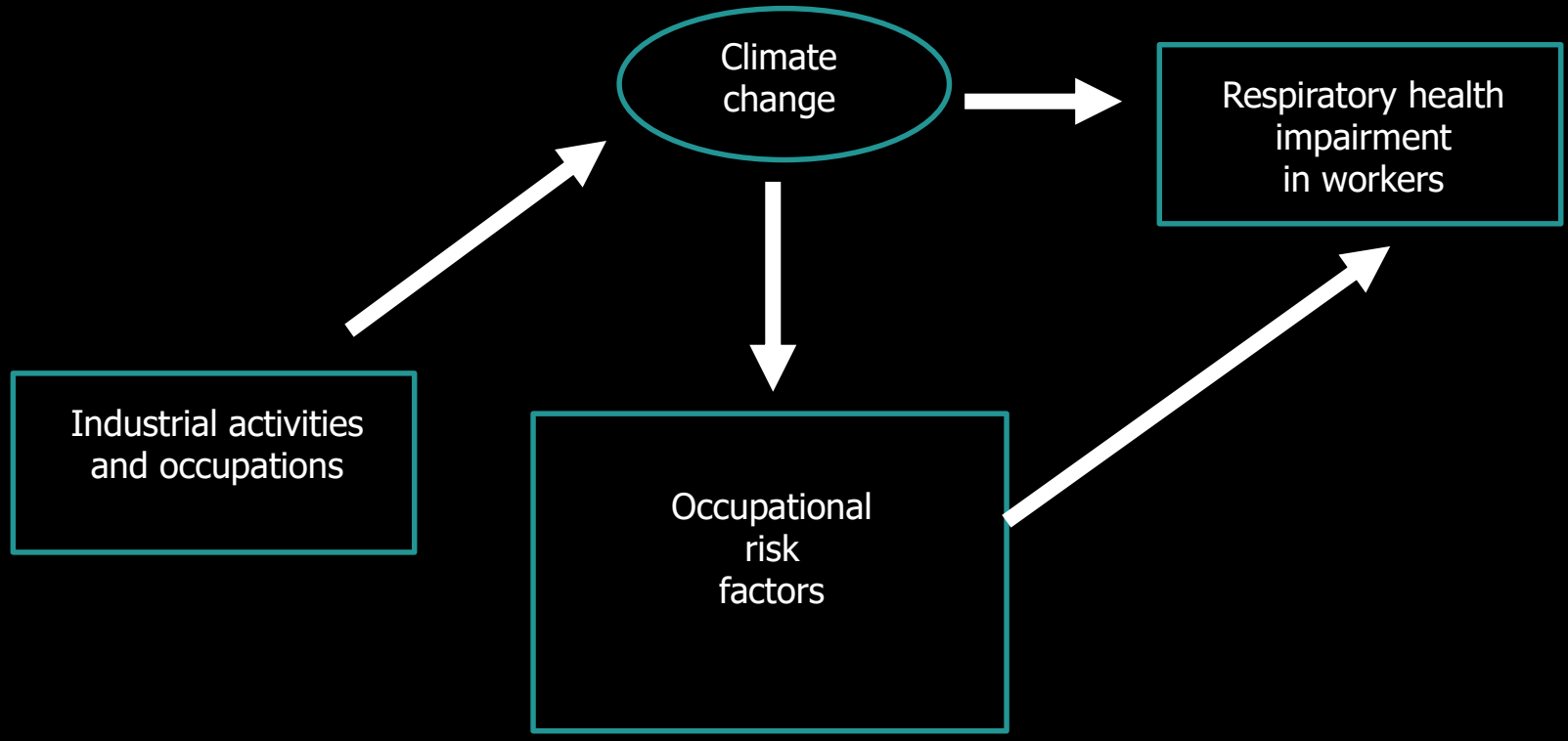
Source / Estimate	Climate-Linked Respiratory Deaths
WHO (2021)	Up to **1.5 million** respiratory deaths/year from air pollution
IPCC (2023)	Projected **tens of thousands of excess respiratory deaths/year** by 2050 under high-emission scenarios
Lancet Countdown (2023)	Climate-sensitive air pollution and heat are among the top **drivers of premature mortality** from respiratory disease worldwide

Trends

- The **Global Burden of Disease (IHME, 2020)** estimates that:
  - Climate change and pollution are **increasing** the burden, especially in LMICs (low- and middle-income countries)
- **Environmental exposures** (air pollution, allergens, occupational dusts/fumes) are key contributors to rising burden.

THE IMPACT OF CLIMATE CHANGE ON WORK-RELATED RESPIRATORY DISEASES

Interrelationship between climate change, risk factors and occupational respiratory health



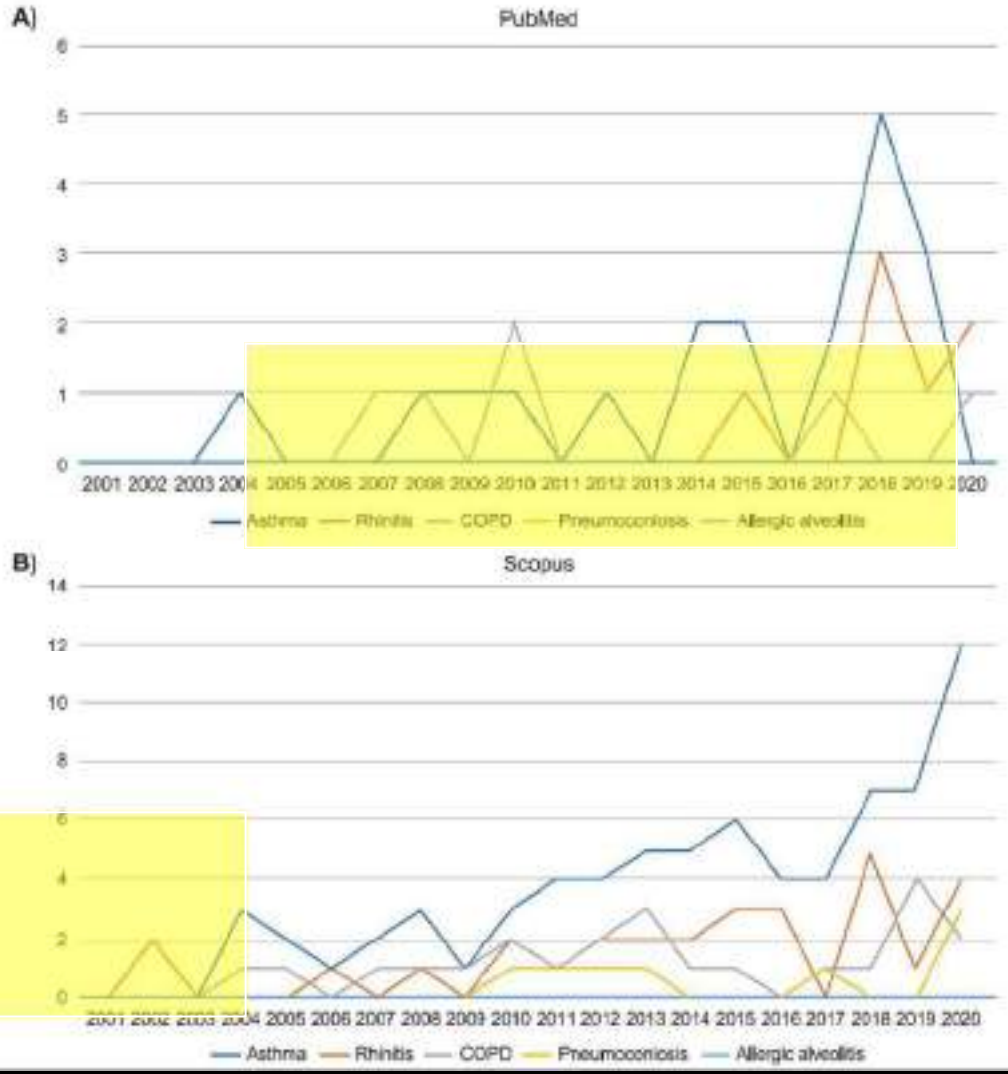
INT J TUBERC LUNG DIS 27(11):858–863

http://dx.doi.org/10.5588/ijtld.23.0131

Non-malignant oc
climate change

M. C. D'Ovidio,[1] A. Lancia,[1]

BACKGROUND: Respiratory
allergic, neoplastic or degenerati
interaction of environmental an
tors, individual susceptibility ar
comorbidities. Asthma and othe
can be worsened by climate cl
other agents in occupational envi
METHODS: PubMed and Scopu
on public and occupational heal
publications and documents on
diseases, asthma, rhinitis, chroni
disease (COPD), pneumoconios
in association with climate chan
RESULTS: Most of the retrie
asthma (75 in Scopus), while th
frequently covered in the scie



Impact of Climate on Workers

•**Climate-Related Occupational Hazards**: Extreme heat, Air pollution, Severe weather events, Natural disasters, Biological hazards (e.g., vector-borne diseases)

• **Amplified Health and Safety Risks**: Increasing exposure intensity and duration, Introducing new, unanticipated hazards, Reducing the predictability of working conditions

• **Compounded Vulnerabilities**:

Migrant workers and those with poor housing or limited socioeconomic support experience:

• Double exposure to climate hazards—both at work and at home

• Higher susceptibility to health effects due to limited access to healthcare and protective infrastructure

⚠️ High-Risk Worker Groups

Worker Group	Climate-Linked Risks
Firefighters	Wildfire smoke, toxic inhalation
Farmers	Dust storms, pesticides, bioaerosols, heat
Construction workers	Ozone, heat, silica dust
Transport workers	Urban air pollution, diesel exhaust
Healthcare & cleanup crews	Mold, disinfection chemicals post-flood
Migrant laborers	Combined home/workplace exposures, poor ventilation

Some figures of the CC occupational burden

Wildfire Smoke Exposure

- **Outdoor workers** (firefighters, farmers, construction): Up to **12–20x higher exposure** to $PM_{2.5}$ during wildfire events compared to the general population.
- In California (2020), wildfire smoke exposure led to:
  - **25% increase in respiratory emergency visits**
  - Estimated **up to 1,200 excess deaths**, a portion linked to occupational exposure.

Air Pollution & Ozone

- **Occupational exposure to ozone** (e.g., outdoor workers in urban areas):
  - Linked to **lung function decline** and **higher asthma and COPD incidence**.
- **WHO (2021)**: Of the **4.2 million deaths annually from air pollution**, a **significant portion occurs in working-age adults**, especially in the informal labor sector.

Some figures of the CC occupational burden

👷 Silica Dust, Drought & Mining

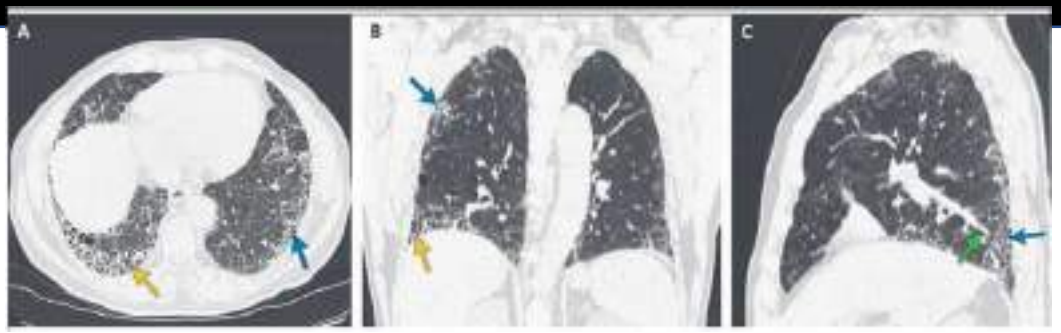
- **Construction and mining workers** face increasing risks from **desertification and drought**, which raise **silica dust levels**:
  - Example: Resurgence of **silicosis** in stone-cutting workers in India and Australia.
  - WHO estimates **up to 40 million workers** globally are at risk of **silica-related respiratory disease**.

🦠 Mold and Biological Hazards

- After climate-linked **flooding or hurricanes**, indoor workers (cleaners, teachers, office staff) face increased exposure to **molds and endotoxins**:
  - Linked to **occupational asthma**, **hypersensitivity pneumonitis**, and **bronchiolitis obliterans**.

Idiopathic pulmonary fibrosis (IPF) stands out as a disease with both occupational and environmental determinants potentially impacted by climate change

A CASE STUDY OF AN ENVIRONMENTAL AND OCCUPATIONAL DISEASE IN A CHANGING CLIMATE: Idiopathic pulomary fibrosis



- Rare progressive chronic interstitial lung disease of complex unknown origin
- Quality of life and prognosis remain very poor with average survival <5 years from diagnosis
- Increasing rates of hospital admissions and deaths due
- to IPF also suggest an increasing burden of disease.
- Identification and elimination of all modifiable causal determinants is ethically imperative to prevent the associated public health burden and individual sufferings

- Raghu G, Remy-Jardin M, Richeldi L, Thomson CC, Inoue Y, Johkoh T, Kreuter M, Lynch DA, Maher TM, Martinez FJ, Molina-Molina M, Myers JL, Nicholson AG, Ryerson CJ, Strek ME, Troy LK, Wijsenbeek M, Mammen MJ, Hossain T, Bissell BD, Herman DD, Hon SM, Kheir F, Khor YH, Macrea M, Antoniou KM, Bouros D, Buendia-Roldan I, Caro F, Crestani B, Ho L, Morisset J, Olson AL, Podolanczuk A, Poletti V, Selman M, Ewing T, Jones S, Knight SL, Ghazipura M, Wilson KC. Idiopathic Pulmonary Fibrosis (an Update) and Progressive Pulmonary Fibrosis in Adults: An Official ATS/ERS/JRS/ALAT Clinical Practice Guideline. Am J Respir Crit Care Med. 2022 May 1;205(9):e18-e47. doi: 10.1164/rccm.202202-0399ST. PMID: 35486072; PMCID: PMC9851481.
- Lederer DJ, Martinez FJ. Idiopathic Pulmonary Fibrosis. N Engl J Med. 2018 May 10;378(19):1811-1823. doi: 10.1056/NEJMra1705751. PMID: 29742380.

GLOBAL BURDEN IPF

TRULY RARE?
NO WORLD DATA!

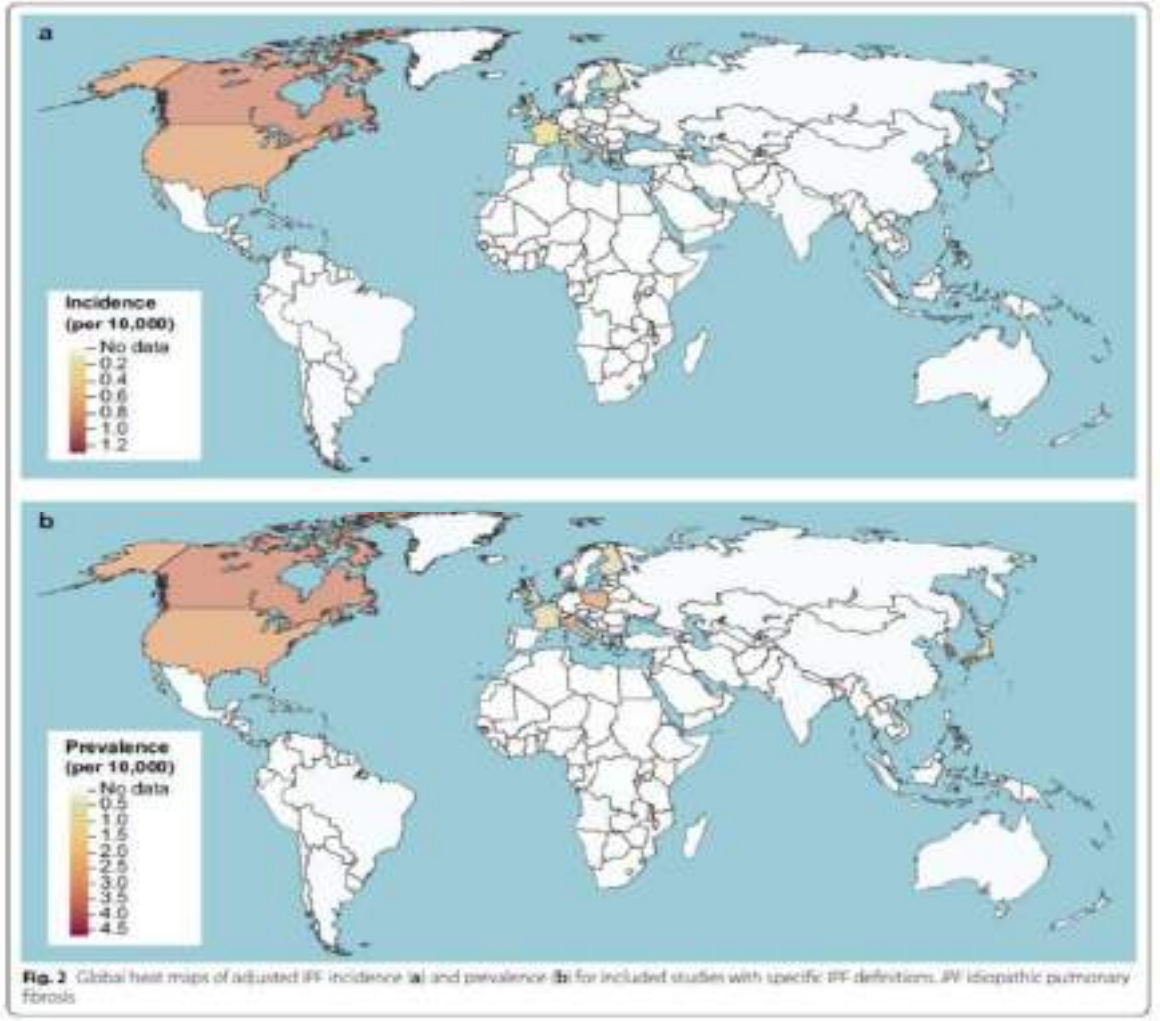


Fig. 2 Global heat maps of adjusted IPF incidence (a) and prevalence (b) for included studies with specific IPF definitions. IPF idiopathic pulmonary fibrosis

Maher TM, Bendstrup E, Dron L, Langley J, Smith G, Khalid JM, Patel H, Kreuter M. Global incidence and prevalence of idiopathic pulmonary fibrosis. Respir Res. 2021 Jul 7;22(1):197. doi: 10.1186/s12931-021-01791-z. PMID: 34233665; PMCID: PMC8261998

NOT SO IDIOPATHIC?

IPF ETIOLOGY: a COMPLEX BLACK BOX

Air pollution

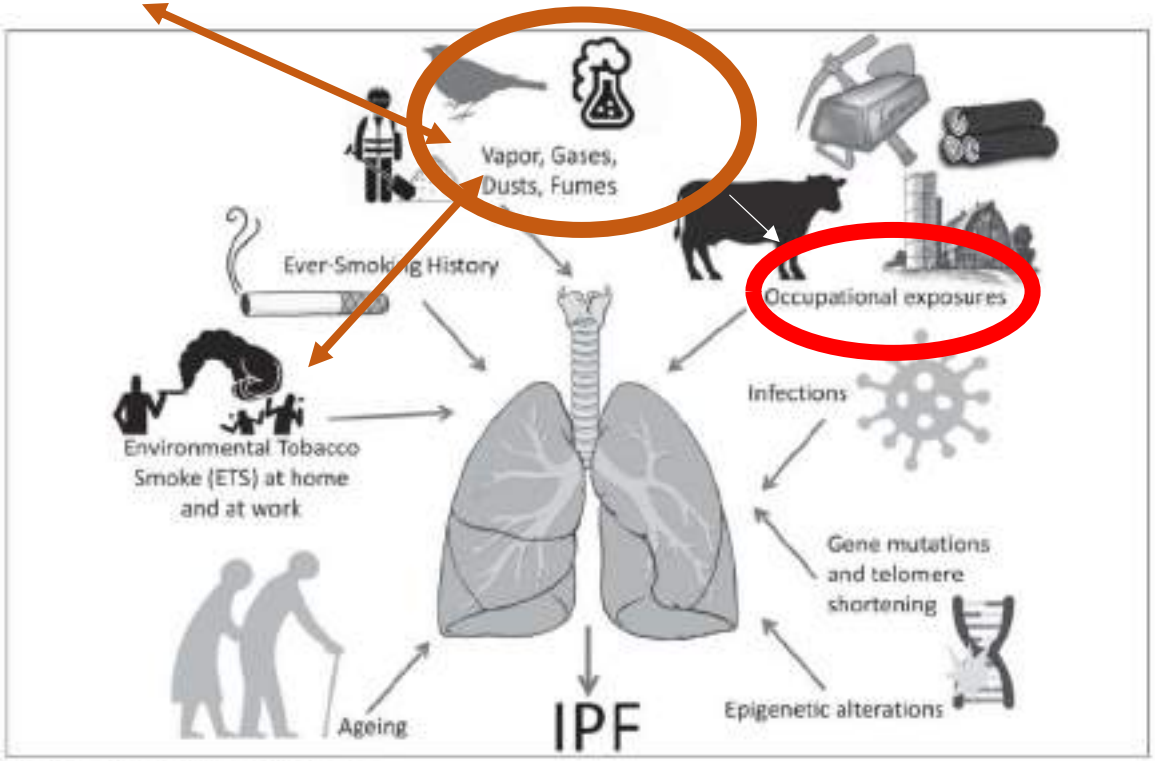


Figure 2 - Risk factors involved in IPF

IPF
Exposome

Ranzieri S, Illica Magrini E, Mozzoni P, Andreoli R, Pelà G, Bertorelli G, Corradi M. Idiopathic pulmonary fibrosis and occupational risk factors. Med Lav. 2019 Dec 17;110(6):407-436. doi: 10.23749/mdl.v110i6.8970. PMID: 31846447; PMCID: PMC7809935.

IPF AND OCCUPATION

IPF: a diagnosis of exclusion?

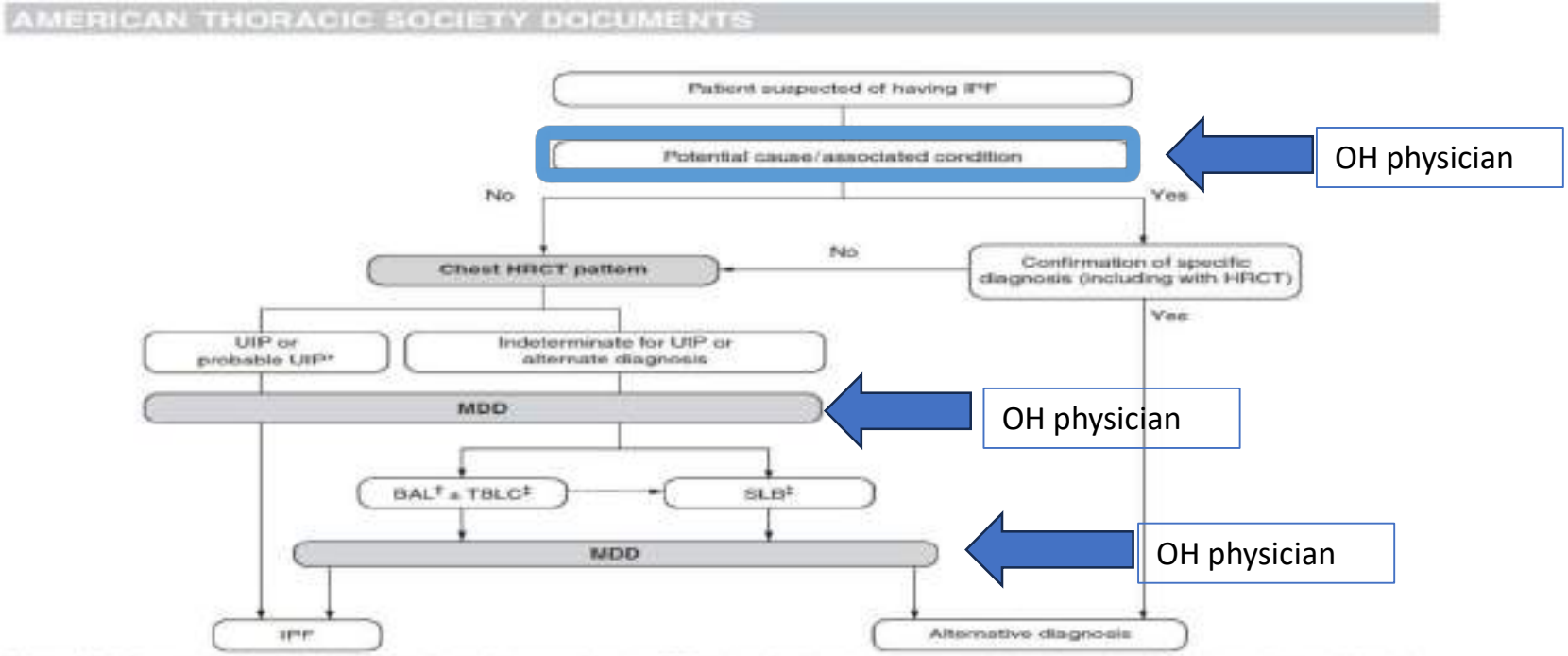


Figure 10. Diagnostic algorithm for idiopathic pulmonary fibrosis (IPF), developed using consensus by discussion. *Patients with a radiological pattern of probable usual interstitial pneumonia (UIP) can receive a diagnosis of IPF after multidisciplinary discussion (MDD) without confirmation by lung biopsy in the appropriate clinical setting (e.g., 60 yr old, male, smoker). BAL may be appropriate in some patients with a probable UIP pattern. †BAL may be performed before MDD in some patients evaluated in experienced centers. ‡Transbronchial lung cryobiopsy (TBLC) may be preferred to surgical lung biopsy (SLB) in centers with appropriate expertise and/or in some patient populations, as described in the text. A subsequent SLB may be justified in some patients with nondiagnostic findings on TBLC. Adapted from Reference 2. HRCT = high-resolution computed tomography.

Raghu G, et a. Idiopathic Pulmonary Fibrosis (an Update) and Progressive Pulmonary Fibrosis in Adults: An Official ATS/ERS/JRS/ALAT Clinical Practice Guideline. Am J Respir Crit Care Med. 2022 May 1;205(9):e18-e47. doi: 10.1164/rccm.202202-0399ST. PMID: 35486072; PMCID: PMC9851481

Carlier, S., Nasser, M., Fort, E. *et al.* Role of the occupational disease consultant in the multidisciplinary discussion of interstitial lung diseases. *Respir Res* **23**, 332 (2022). https://doi.org/10.1186/s12931-022-02257-6.

Work-related interstitial lung disease: what is the true burden?

Authors: De Matteis, S. [1]; Murgia, N. [2];
Source: The International Journal of Tuberculosis and Lung Disease. Volume 26. Number 11. 1 November 2022.
Publisher: International Union Against Tuberculosis and Lung Disease
DOI: https://doi.org/10.5588/ijtld.22.0212

< previous article view table of contents next article >

Abstract References Citations Supplementary Data Suggestions

Interstitial lung diseases (ILDs) include a large variety of fibrotic lung conditions caused by genetic and environ
Occupational exposures might also play a significant role, but the real health burden is currently unknown. He
role of work-related exposures in ILDs, focussing on idiopathic pulmonary fibrosis (IPF) and hypersensitivity pr
performed a focused review of the literature on work-related HP and IPF over the past 5 years. Using a meta-a
quantified the occupational burden of IPF and HP, and estimated that occupational exposures to metal, silica a
smoke increased IPF risk with a pooled odds ratio of 1.7 (95% CI 1.42–2.03). The proportion of HP cases relate
was 17% (95% CI 7–28). Our review supports the hypothesis that occupational exposures are a significant risk
aetiopathogenesis of IPF and HP. We recommend that further research be performed to identify the underlyin
the maximum permitted exposure to reduce the associated IPF and HP burden.

OCCUPATIONAL RISK FACTORS

TABLE 1 Overt or occult environmental risk factors for idiopathic pulmonary fibrosis

	Risk factor
Intrinsic factors	Gastro-oesophageal reflux
	Microaspiration
	Microbiome
	Viral infection
Extrinsic factors	
Domestic/environmental	Tobacco smoke
	Wood fires
	Birds (including poultry, bird droppings, birdfeeders)
	Feather products (including feather duvets, comforter, pillows, jackets)
	Moulds (visible or unseen)
	Organic dusts
	Ventilation
	Hairspray
	Air pollution
Occupational[#]	Welding
	Farming/agriculture
	Hairdressing
	Dentists/dental technicians
	Metal dust
	Wood dust/paper mill factory workers
	Livestock, particularly birds
	Nuclear waste/radiation hazards
	Chemicals
	Aluminium, Corien®
	Stone cutting/sand/granite/silica
	Talc

[#]: these and other occupational exposures may be the cause of "occupational lung diseases" and thus not truly risk factors for patients diagnosed with idiopathic pulmonary fibrosis (IPF). Nevertheless, these may be occult if the patient otherwise diagnosed with IPF is not gainfully engaged with such environmental factors/exposures.

Occupational exposures more frequently associated to IPF in epidemiological studies:

- VGDF (vapour, gas, dust, fibres)
- Crystalline Silica
- Abestos
- Organic dusts
- Metals
- Wood dusts
- Agriculture
- ETS (Environmental tobacco smoking)

Reported up to 60% prevalence of occupational exposure among IPF cases in tertiary hospital centres

- Sack C, Raghu G. Idiopathic pulmonary fibrosis: unmasking cryptogenic environmental factors. Eur Respir J. 2019 Feb21;53(2):1801699. doi: 10.1183/13993003.01699-2018. PMID: 30487201.
- Carlier, S., Nasser, M., Fort, E. et al. Role of the occupational disease consultant in the multidisciplinary discussion of interstitial lung diseases. Respir Res 23, 332 (2022). https://doi.org/10.1186/s12931-022-02257-6

OCCUPATIONAL IPF RISK ESTIMATES

VGDF

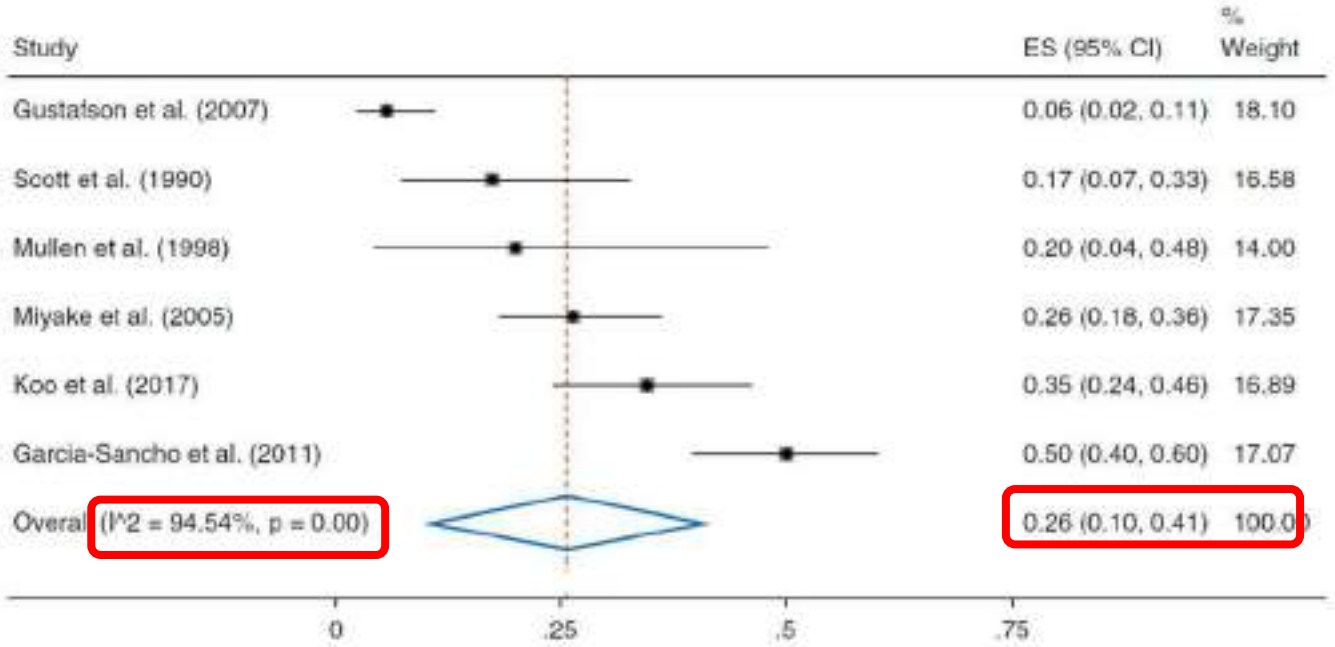


Figure 4. Idiopathic pulmonary fibrosis (IPF): population attributable fraction (PAF) from vapors, gas, dust, or fumes (VGDF). Forest plot of studies relevant to estimating the occupational contribution to IPF of VGDF (combined categories of exposure considered in the studies included). The estimated PAF, confidence interval (CI), and weighted contribution for each study are shown, as well as the calculated pooled estimate (red dashed line) and 95% CI. For IPF, the pooled PAF for VGDF is 26% (95% CI, 10–41%). ES = effect size.

Blanc PD, Annesi-Maesano I, Balmes JR, Cummings KJ, Fishwick D, Miedinger D, Murgia N, Naidoo RN, Reynolds CJ, Sigsgaard T, Torén K, Vinnikov D, Redlich CA. The Occupational Burden of Nonmalignant Respiratory Diseases. An Official American Thoracic Society and European Respiratory Society Statement. Am J Respir Crit Care Med. 2019 Jun 1;199(11):1312-1334. doi: 10.1164/rccm.201904-0717ST. PMID: 31149852; PMCID: PMC6543721.

OCCUPATIONAL IPF risk estimates

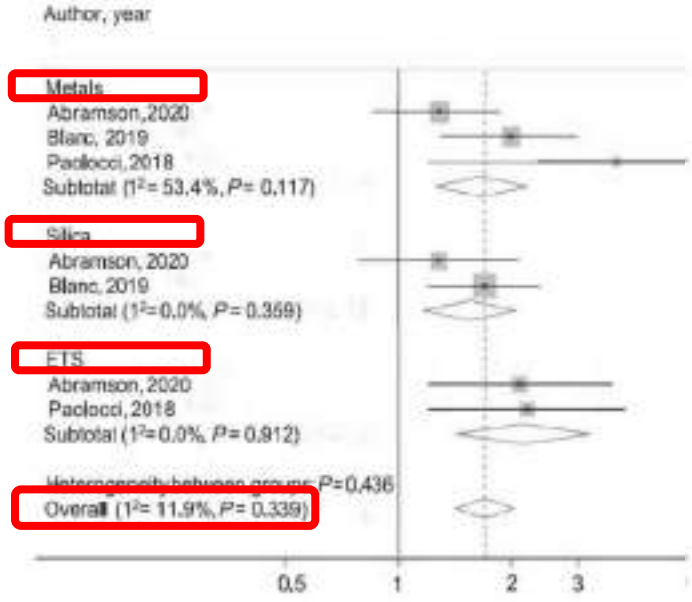
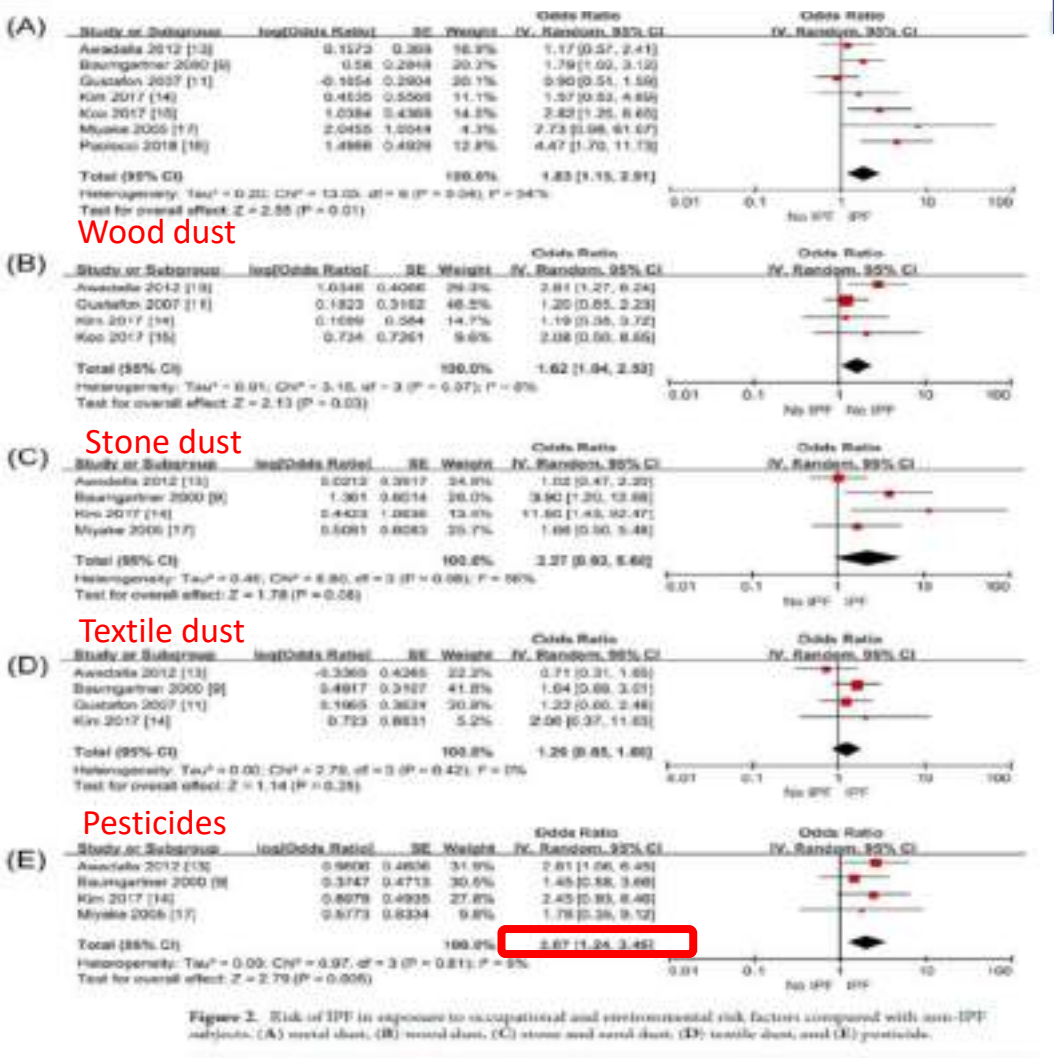


Figure 1 Pooled risk estimates (ORs and 95% CIs) of work-related IPF by se
pooled estimate. OR = odds ratio; ETS = environmental tobacco smoking; CI =

- De Matteis S, Murgia N. Work-related interstitial lung disease: what is the true burden? Int J Tuberc Lung Di
10.5588/ijtld.22.0212. PMID: 36281049.
- Park Y, Ahn C, Kim TH. Occupational and environmental risk factors of idiopathic pulmonary fibrosis: a syste
2;11(1):4318. doi: 10.1038/s41598-021-81591-z. PMID: 33654111; PMCID: PMC7925580.

Metals



OCCUPATIONAL IPF ESTIMATES

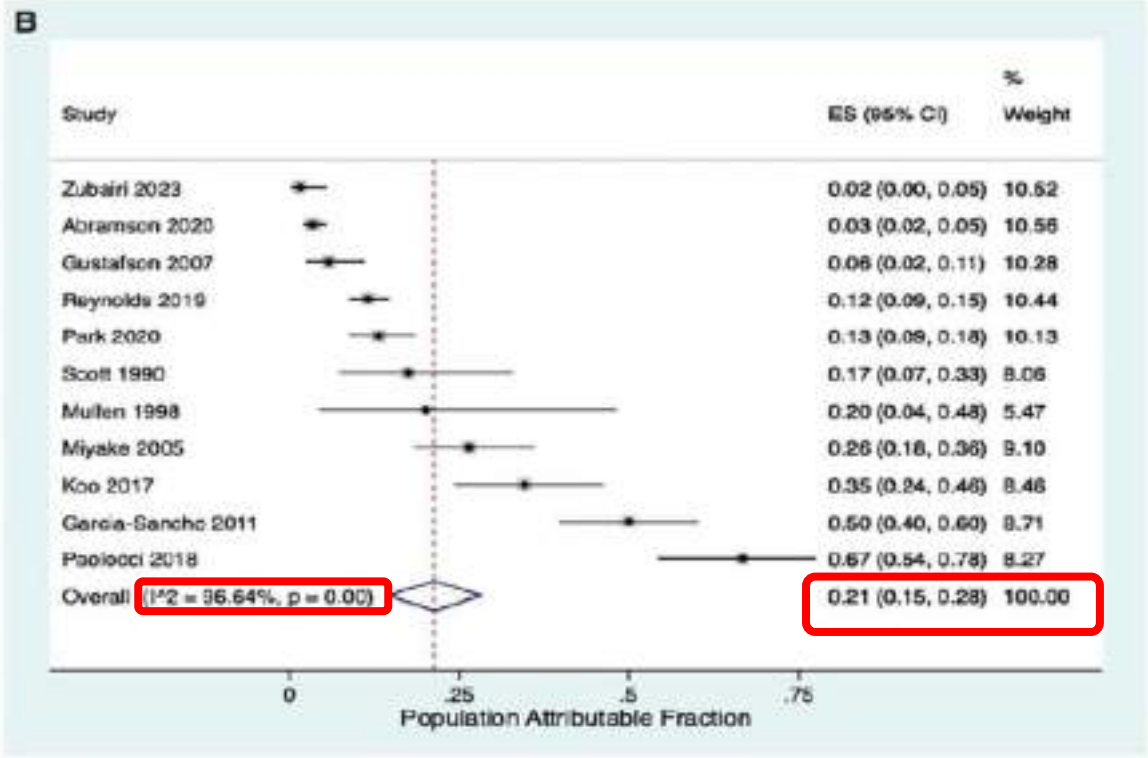


Figure 3. Idiopathic pulmonary fibrosis (IPF) odds ratio (OR) and population attributable fraction (PAF) for vapors, gas, dust, or fumes from case-control studies. Forest plot of articles relevant to estimating the occupational contribution to IPF of vapors, gas, dust, or fumes (combined categories of exposure considered in the studies included). The estimated OR (*A*) and PAF (*B*), confidence interval (CI), and weighted contribution for each study are shown, as are the calculated pooled estimate (red dashed line) and 95% CI. (*A*) The pooled OR is 1.9 (95% CI, 1.4–2.6). (*B*) The pooled PAF is 24% (95% CI, 16–32%). ES = effect size.

Gandhi SA, Min B, Fazio JC, Johannson KA, Steinmaus C, Reynolds CJ, Cummings KJ. The Impact of Occupational Exposures on the Risk of Idiopathic Pulmonary Fibrosis: A Systematic Review and Meta-Analysis. Ann Am Thorac Soc. 2024 Mar;21(3):486-498. doi: 10.1513/AnnalsATS.202305-402OC. PMID: 38096107; PMCID: PMC10913770.

OCCUPATIONAL IPF risk estimates

- In a population-based case-control study of 503 cases 902 controls in Australia
- Increased IPF risk for occupational exposures to:

Table 3 Associations with occupational asbestos exposure assessed by AsbJEM: ORs, 95% CIs and p values

Asbestos exposure			Unadjusted ORs	Adjusted ORs			
Quartiles		Fibre-years/mL		OR*	95% CI	P value	OR†
Reference group	Q1	≤0.00295	1	1			1
	Q2	0.00296, ≤0.00425	1.20	1.21	(0.88 to 1.66)	0.233	1.2
	Q3	0.00426, ≤0.06255	1.39	1.41	(1.03 to 1.93)	0.032	1.4
	Q4	0.06256 to 8.256	1.55	1.57	(1.15 to 2.15)	<0.001	1.61

*Adjusted for age, sex, state and tobacco smoking (never/past/current).
†Additionally adjusted for age, sex, state, tobacco smoking (never/past/current) and pack-years.
AsbJEM, asbestos Job Exposure Matrix.

Table 4 Occupational asbestos exposure, smoking, genotype and IPF; interaction terms and stratified analysis (genotyped participants only, n=902)

Exposure*	Adjusted OR† (95% CI; p value)
Interaction model (asbestos*smoking)	
Ever asbestos exposed	0.6 (0.4 to 1.1; 0.08)
Ever smoked	0.9 (0.6 to 1.5; 0.68)
Ever asbestos exposed and ever smoked interaction	2.2 (1.2 to 4; 0.01)
Interaction model (asbestos*genotype)	
Ever asbestos exposed	1.0 (0.7 to 1.5; 0.84)
Genotype	3.7 (2.4 to 5.8; 0.001)
Ever asbestos exposed and genotype	1.3 (0.8 to 2.3; 0.3)
Interaction model (smoking*genotype)	
Ever smoked	1.29 (0.86 to 1.92; 0.22)
Genotype	3.95 (2.33 to 6.69; 0.001)
Ever smoked and genotype	1.15 (0.62 to 2.13; 0.65)
Stratified interaction model (asbestos*smoking)	
GT or TT	
Ever asbestos exposed	0.4 (0.2 to 1; 0.06)
Ever smoked	0.6 (0.3 to 1.3; 0.2)
Ever asbestos exposed and ever smoked interaction	5.4 (1.8 to 15.6; 0.002)

*Ever asbestos exposed was defined as ever having had a high or medium asbestos exposure risk job, defined on the basis of occupational proportional mortality ratios for mesothelioma.[16] Genotype of *MUC5B* rs35705950, T is the minor allele.
†Adjusted for age±smoking, smoking was not adjusted for when it was considered as an exposure. Centre was not adjusted for in this analysis because numbers were too small for one centre. Analysis limited to the 20 centres which did have sufficient numbers showed that adjusting for centre did not significantly change our results.
IPF, idiopathic pulmonary fibrosis.

- Abramson MJ, Murambadoro T, Alif SM, Benke GP, Dharmage SC, Glaspole I, Hopkins P, Hoy RF, Klebe S, Moodley Y, Rawson S, Reynolds Australia: case-control study. Thorax. 2020 Oct;75(10):864-869. doi: 10.1136/thoraxjnl-2019-214478. Epub 2020 Jul 13. PMID: 32660982.
- Reynolds CJ, Sisodia R, Barber C, Moffatt M, Minelli C, De Matteis S, Cherrie JW, Newman Taylor A, Cullinan P. What role for asbestos in 10.1136/oemed-2022-108404. Epub 2023 Jan 12. PMID: 36635100; PMCID: PMC9887381.

WORSE PROGNOSIS FOR OCCUPATIONAL IPF?

In a large IPF cohort in the nationwide IPF Registry of Korea occupational exposure to wood and stone dusts were associated with higher IPF mortality

Table 6. Cox regression analyses of risk factors for mortality

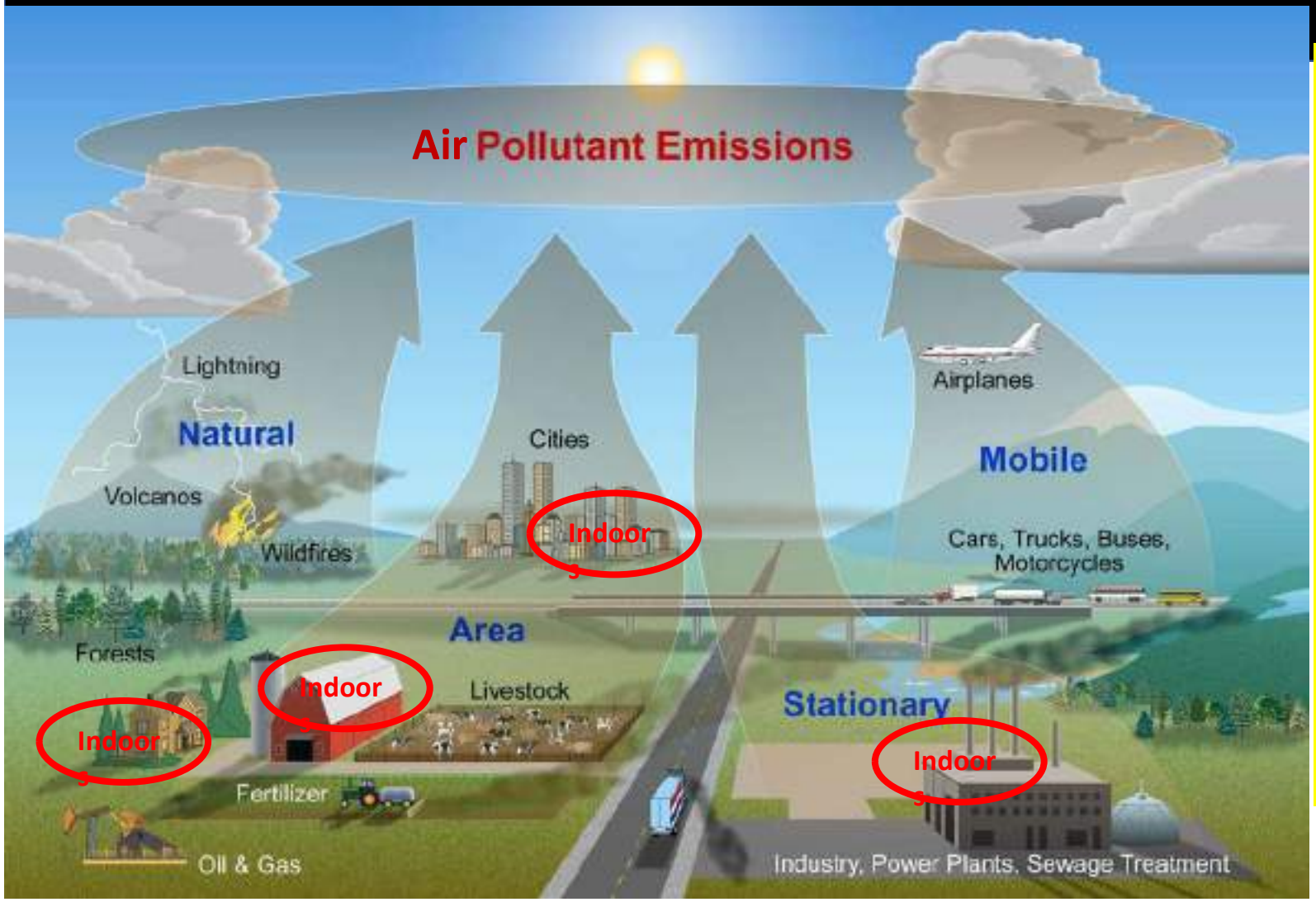
	Univariate analysis			Multivariate analysis		
	HR	CI	p-value	HR	CI	p-value
Age	1.038	1.014–1.063	0.002	1.046	1.007–1.086	0.021
Sex	1.210	0.766–1.911	0.414	-	-	-
Smoking			0.489			
Nonsmoker	1					
Ex-smoker	0.517	0.210–1.271		-	-	-
Never smoker	0.634	0.313–1.288		-	-	-
Six-minute walking test						
Walking distance	0.997	0.995–0.998	<0.001	0.999	0.995–1.002	0.435
Minimum saturation	0.945	0.919–0.972	<0.001	0.949	0.918–0.980	0.003
Pulmonary function test						
FVC	0.969	0.957–0.981	<0.001	0.999	0.977–1.022	0.956
DLco	0.965	0.954–0.975	<0.001	0.996	0.979–1.014	0.685
Initial CT pattern			0.160			
Inconsistent with UIP	1	-	-			
Probable UIP	2.393	0.746–7.675	0.142	-	-	-
UIP	2.861	0.894–9.161	0.077	-	-	-
Honeycombing	1.459	0.961–2.217	0.076	-	-	-
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Antifibrotics	2.037	1.328–3.126	0.001	0.497	0.262–0.944	0.033
Acute exacerbation	5.392	3.698–7.859	<0.001	3.703	2.165–6.332	<0.001
Clinical trial participation	0.723	0.293–1.789	0.483	-	-	-
Exposure						
Chemicals	1.269	0.715–2.310	0.402	-	-	-
Wood dust	2.099	1.089–4.048	0.027	2.449	1.094–5.484	0.029
Metal dust	3.772	0.929–15.316	0.063	-	-	-
Fabric dust	0.845	0.287–2.675	0.775	-	-	-
Stone dust	2.037	1.242–3.339	0.005	2.269	1.229–4.204	0.009
Family history of ILD	0.587	0.186–1.853	0.364	-	-	-

HR: hazard ratio; CI: confidence interval; FVC: forced vital capacity; DLco: diffusing capacity for carbon monoxide; CT: computed tomography; UIP: usual interstitial pneumonia; GAP: gender-age-physiology; ILD: interstitial lung disease.

Jegal Y, Park JS, Kim SY, Yoo H, Jeong SH, Song JW, Lee JH, Lee HL, Choi SM, Kim YW, Kim YH, Choi HS, Lee J, Uh ST, Kim TH, Kim SH, Lee WY, Kim YH, Lee HK, Lee EJ, Heo EY, Yang SH, Kang HK, Chung MP; Korea ILD Study Group. Clinical Features, Diagnosis, Management, and Outcomes of Idiopathic Pulmonary Fibrosis in Korea: Analysis of the Korea IPF Cohort (KICO) Registry. Tuberc Respir Dis (Seoul). 2022 Apr;85(2):185-194. doi: 10.4046/trd.2021.0123. Epub 2021 Dec 13. PMID: 34902237; PMCID: PMC8987666.

Main limit: retrospective

IPF AND AIR POLLUTION



Main air pollutants:

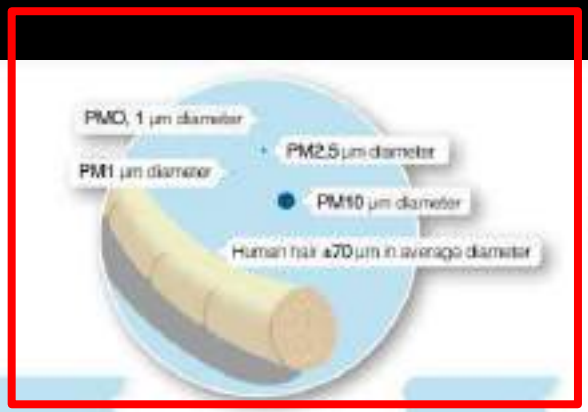
- Gases: NO_2, SO_2, CO, O_3, VOCs, etc.

- PM (Particulate Matter) (size and composition)

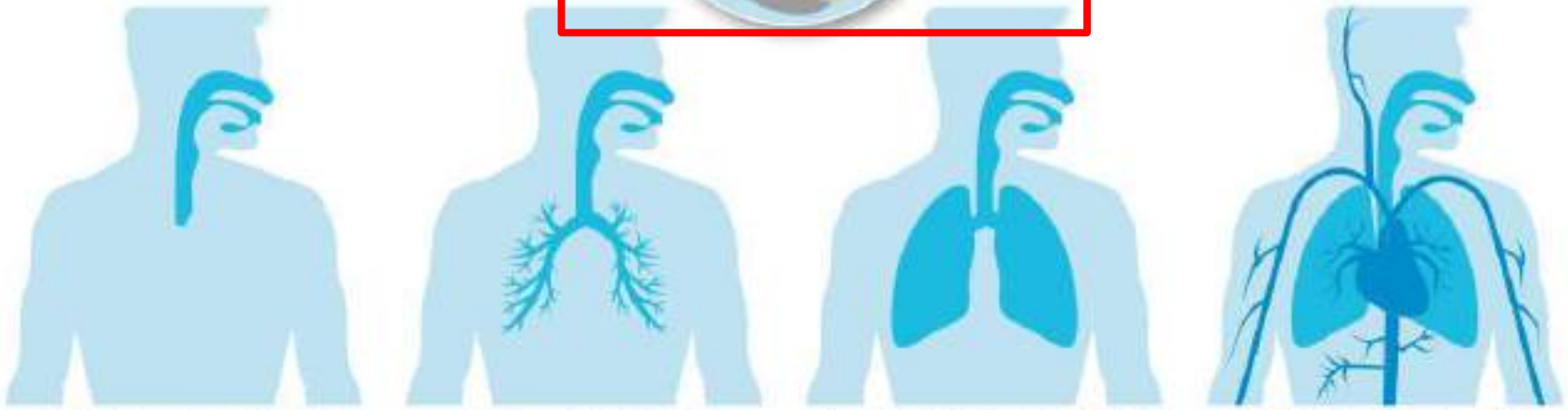
PM the big killer

Penetration of particles into the body

(The smaller the particles, the more dangerous they are)



Particulate matter, smaller than a human hair



COARSE PARTICLES
Upper respiratory tract
Size = < 10 µm

PM10 = 0.01mm
- pollen
- desert dust

FINE PARTICLES
Lower respiratory tract
Size = < 2.5 µm

PM2.5 = 0.0025mm
- bacteria
- fungal and mold spores
- toner dust

INHALEABLE PARTICLES
Aveoli
Size = < 1 µm

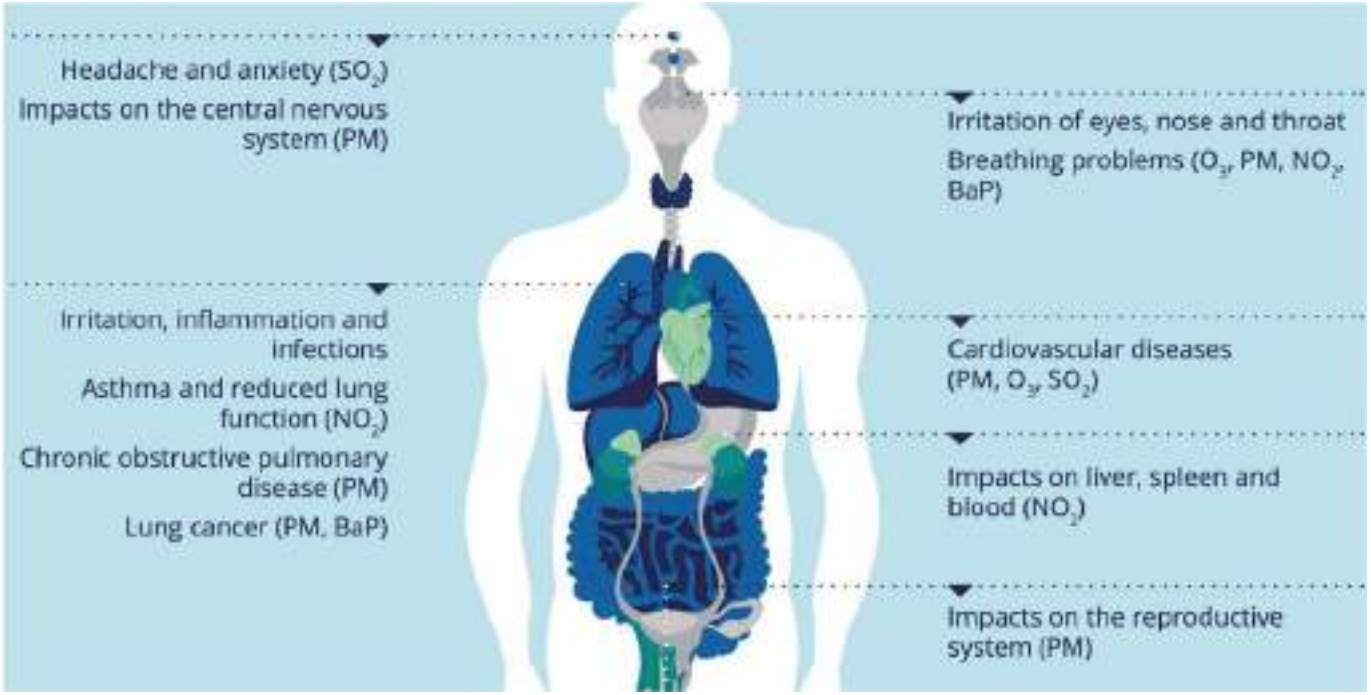
PM1 = 0.001 mm
- viruses
- exhaust gases

ULTRAFINE PARTICLES
Bloodstream/whole body
Size = < 0.1 µm

PM0.1 = 0.0001mm
- Nano particules

Both PM size and composition are of concerns

The various impacts of air pollution



- **Health**
- **Environment**: biodiversity, crops
- **Climate change**
- **Built** environment
- **Economic** costs

Further information: [WHO webpage]

CLIMATE CHANGE-RELATED AIR POLLUTION TRENDS

- **Virtually certain**
  - **Increased extended pollen seasons and pollen production and allergenicity and due to**
    - Increasing temperatures
    - Plants moves
  - **Increased outdoor ozone level due to**
    - Increasing temperatures
    - Windiness and stagnant air conditions
    - Urbanization
    - Natural sources of air pollutant emissions (→biogenic VOCs)
  - **Increased outdoor PM level due to**
    - Increasing emissions from
      - fossil fuel-fired power plants due to demand for electricity for cooling (due to temperature increase)
      - Urbanization and traffic
    - Increasing natural sources of air pollutant emissions
      - Wildfire smoke induced by drought and heat
      - Desertification → Sand storm

Desertification

Sand storm

Wildfire

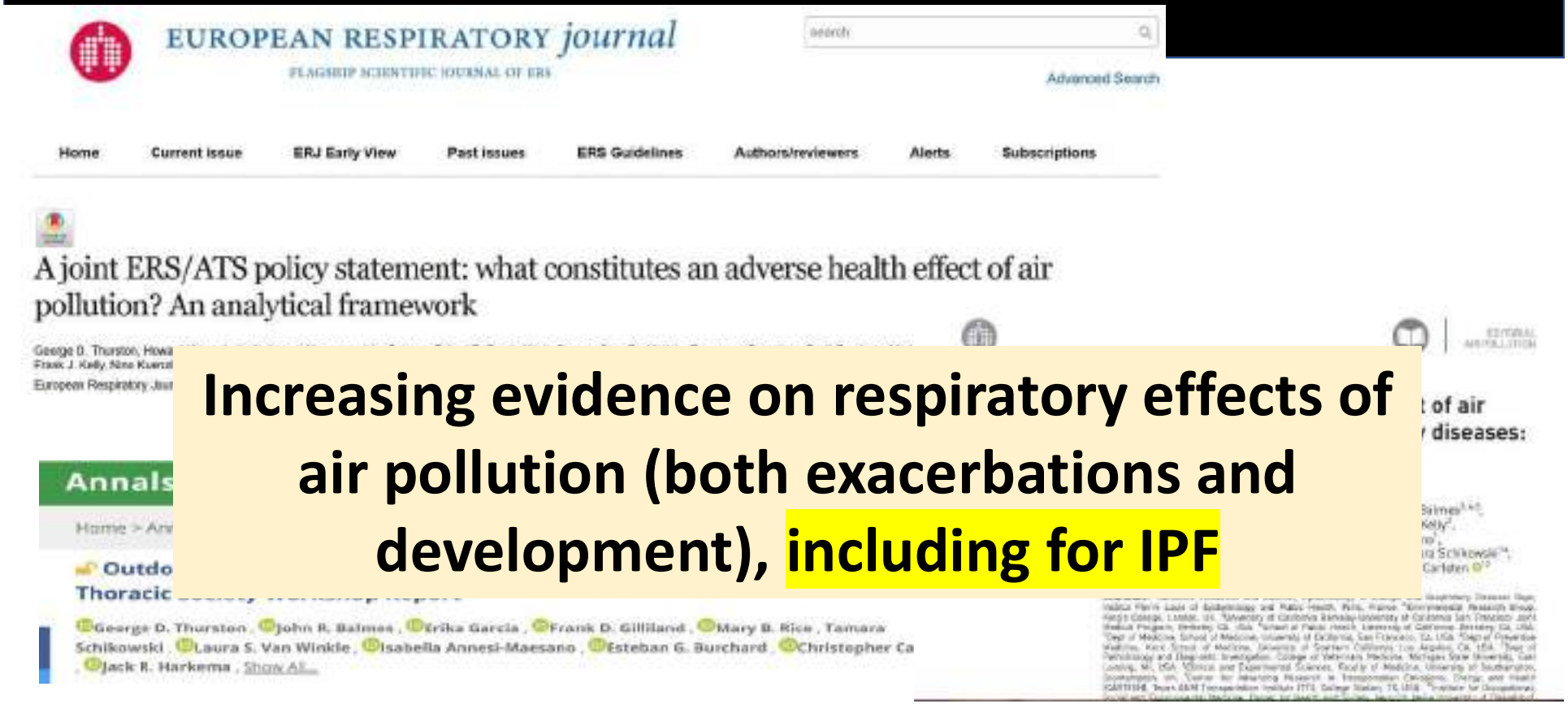
Urbanization and trafic

Industry in developing countries

- **To be further confirmed**
  - Increase of indoor air pollutants

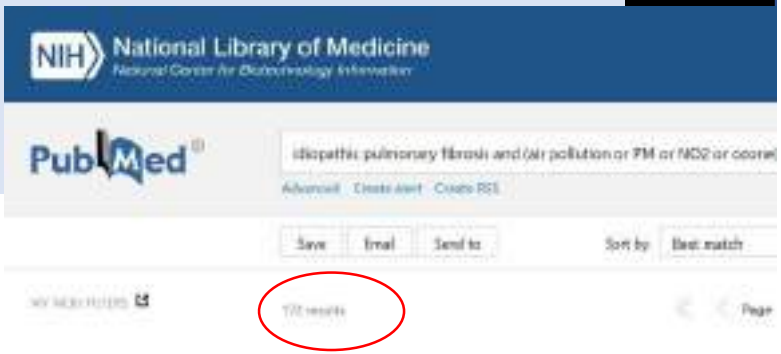
Natural disasters and respiratory health

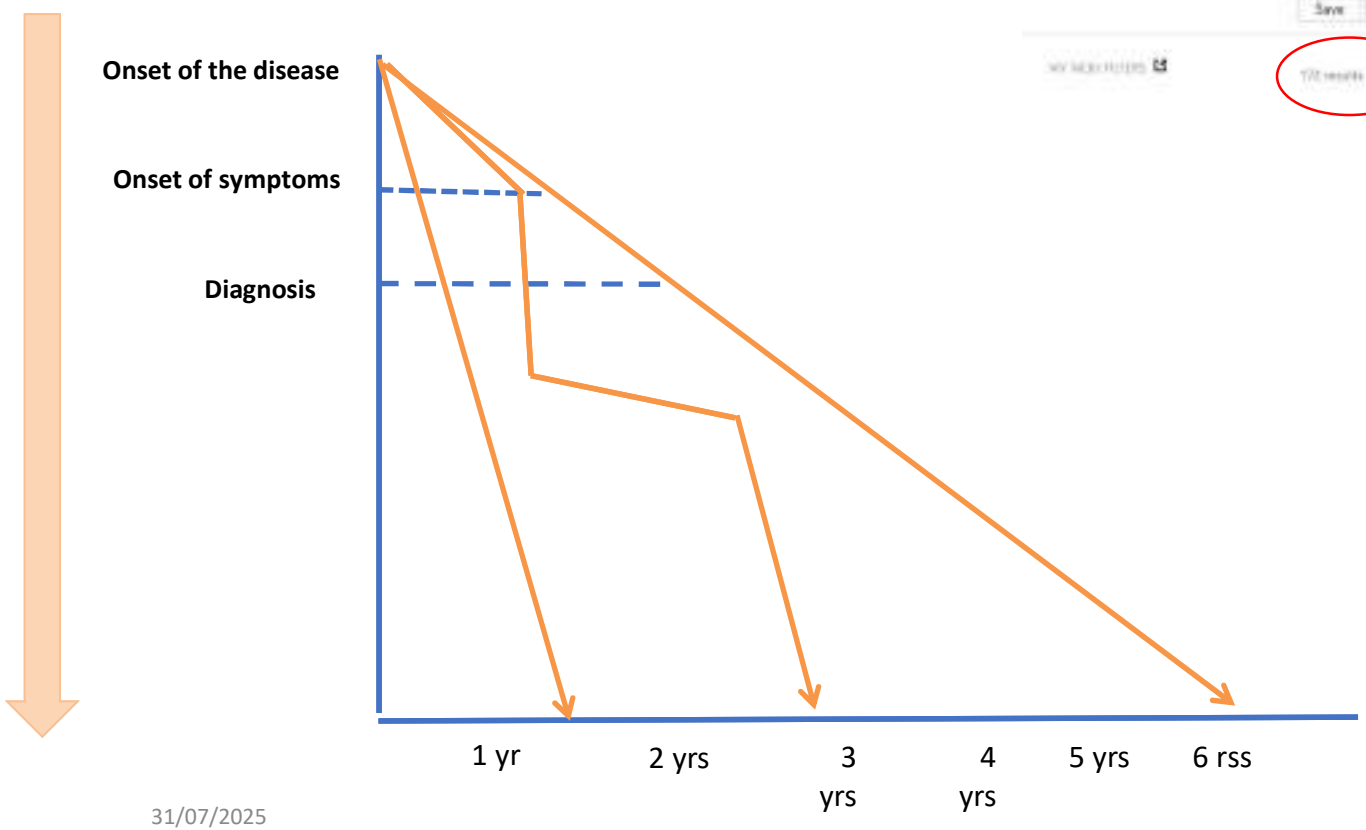
Isabella Annesi-Maesano[1,2], Hasan Bayram[3,4], Lorenzo Cecchi[5], Daniel Croft[6], Gennaro D'Amato, Arundhati Garud[8,9], Ozgecan Kayalar[3,4], Mehdi Misraedi[10], Subhabrata Moitra[9], Vanitha Sampath[11], Neeta Thakur[6], Kari Nadeau[11], John Balmes[12,13]



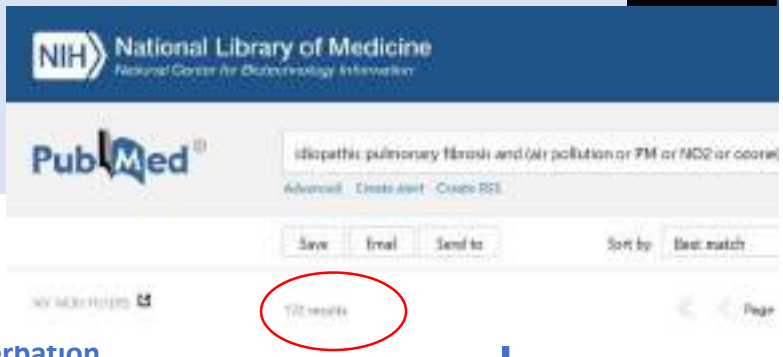
Increasing evidence on respiratory effects of air pollution (both exacerbations and development), including for IPF

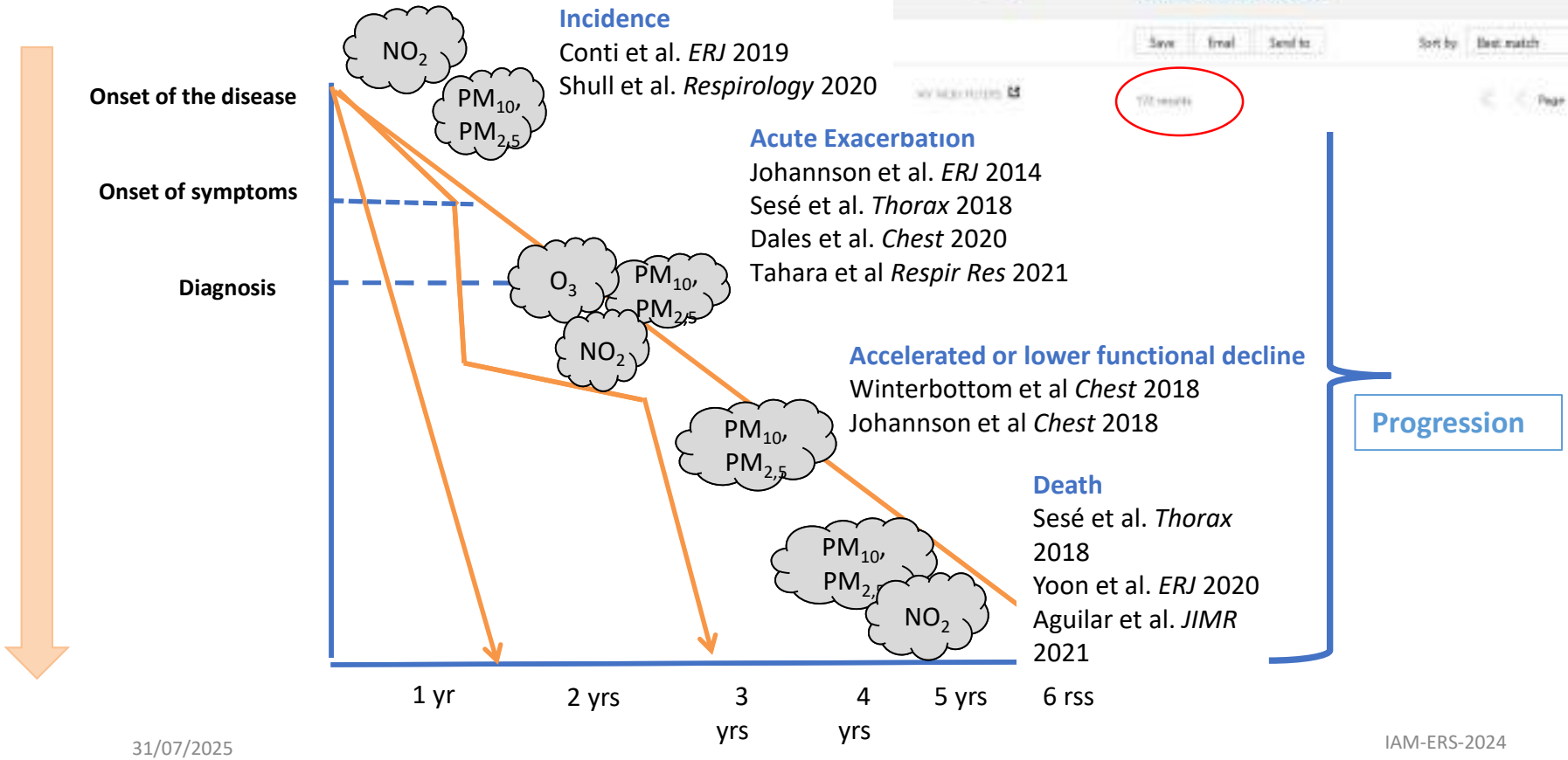
Air Pollution and IPF





Air Pollution and IPF





Incidence
Conti et al. *ERJ* 2019
Shull et al. *Respirology* 2020

Acute Exacerbation
Johannson et al. *ERJ* 2014
Sesé et al. *Thorax* 2018
Dales et al. *Chest* 2020
Tahara et al *Respir Res* 2021

Accelerated or lower functional decline
Winterbottom et al *Chest* 2018
Johannson et al *Chest* 2018

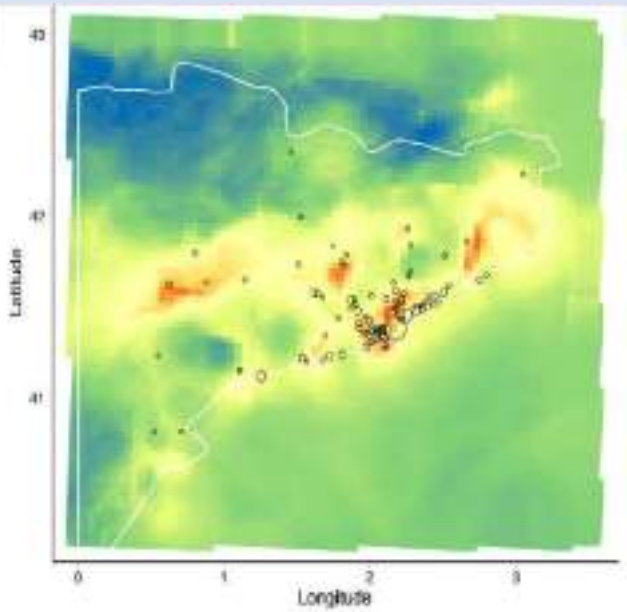
Death
Sesé et al. *Thorax* 2018
Yoon et al. *ERJ* 2020
Aguilar et al. *JIMR* 2021

Progression

IPF development (incidence?) and air pollution

+ 8% Incidence (10 NO_2 µg/m^3)

Conti et al. *ERJ* 2017



$PM_{2,5}$

From the last 10 yrs using CALIOPE modeling

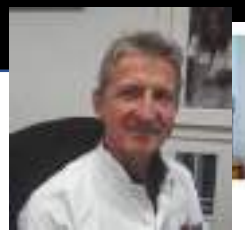
Regions of notable air pollution juxtaposed with higher prevalence of idiopathic pulmonary fibrosis (IPF).

Shull et al. *Respirology* 2020

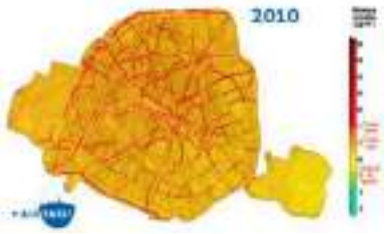

Thorax Online First, published on August 15, 2017 as 10.1136/thoraxjnl-2017-208967

ORIGINAL ARTICLE

Role of atmospheric pollution on the natural history of idiopathic pulmonary fibrosis



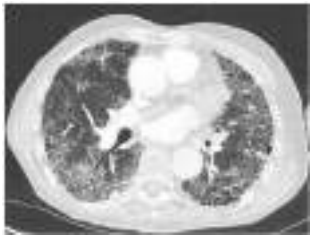
French Cohort of FIbrosis (COFI)
(n=196)



Short-Term Air pollution exposure and IPF acute exacerbations

Exposition	Augmentation	HR* (Conf Int$_{95\%}$)	p-value
O_3	10 µg/m^3	1,26 (1,01 – 1,58)	0,045*
NO_2	10 µg/m^3	0,92 (0,73 – 1,18)	0,509
PM_{10}	10 µg/m^3	0,78 (0,53 – 1,14)	0,197
$PM_{2.5}$	10 µg/m^3	1,04 (0,58 – 1,87)	0,902

6-week exposure



*HR=Hazard ratio

Long-Term Air pollution exposure and IPF mortality

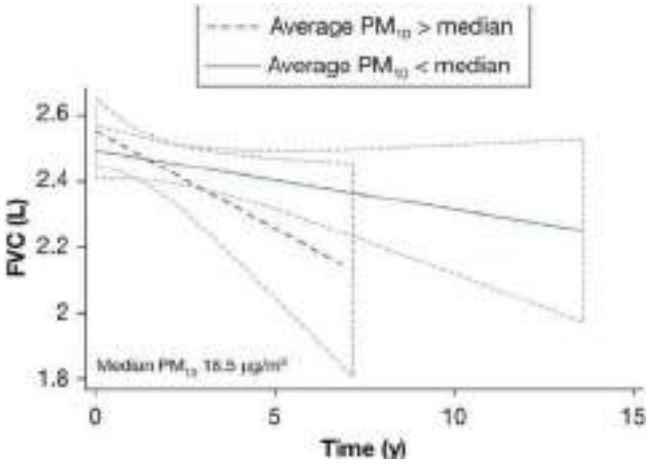
Exposure	Increase	HR* (Conf Int$_{95\%}$)	p-value
O_3	10 µg/m^3	0,89 (0,66 -1,18)	0,43
NO_2	10 µg/m^3	1,01(0,79 – 1,29)	0,90
PM_{10}	10 µg/m^3	2,01 (1,07 – 3,77)	0,03
$PM_{2.5}$	10 µg/m^3	7,93 (2,93 – 21,33)	<0,001

2005 WHO AQS: 100% of patients $PM_{2.5}$

Sesé *Thorax 2018*

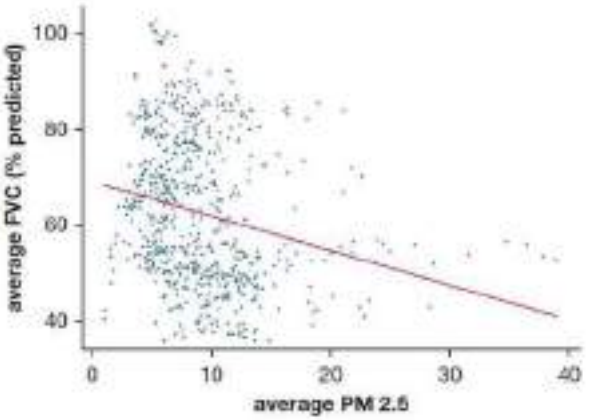
IPF functional decline and air pollution

238 patients



-46 cc/yr of FVC (p = 0,008) per **5 µg/m³ of PM_{10}**

Winterbottom C et al. *Chest* 2018



25 patients

Air Pollution Exposure Is Associated With Lower Lung Function, but Not Changes in Lung Function

Johannson K. et al *Chest* 2018

JAMA Internal Medicine | Original Investigation

Association of Particulate Matter Exposure With Lung Function and Mortality Among Patients With Fibrotic Interstitial Lung Disease

Gillian C. Goobie, MD, PhD; Christopher Carlsten, MD, MPH; Kerri A. Johannson, MD, MPH; Naseem Khalil, MD; Veronica Marcoux, MD; Deborah Assayag, MD; Hélène Manganas, MD; Jolene H. Fisher, MD; Martin R. J. Kolb, MD, PhD; Kathleen O. Lindell, RN, PhD; James P. Fabisiak, PhD; Xiaoping Chen, MS; Kevin F. Gibson, MD; Yingze Zhang, PhD; Daniel [redacted]; Christopher J. Ryerson, MD, MAS; S. Mehdi Nouraie, MD, PhD

2022

6,683 IPF patients
In Canadian and
USA registers

- $PM_{2.5}$ associated with worse lung function and mortality or transplant among some but not all patients with ILD.
- Sulfate, ammonium, and black carbon associated with the most potential risk

	Simmons cohort (n = 1424)		PFF cohort (n = 1870)		CARE-PF cohort (n = 3389)		Combined cohorts meta-analysis (n = 6683)	
Pollutant	HR (95% CI)	P value	HR (95% CI)	P value	HR (95% CI)	P value	HR (95% CI)	P value
$PM_{2.5}$	1.05 (1.01-1.09)	.02[b]	1.02 (0.98-1.06)	.38	0.98 (0.96-1.01)	.16	1.09 (1.05-1.14)	<.001[b]
SO_4^{2-}	1.21 (1.09-1.35)	<.001[b]	1.27 (1.08-1.50)	.005[b]	0.33 (0.26-0.43)	<.001[b]	2.11 (1.81-2.47)	<.001[b]
NO_3^-	0.77 (0.63-0.94)	.01[b]	0.94 (0.81-1.09)	.42	0.81 (0.66-1.00)	.048[b]	0.99 (0.79-1.24)	.94
NH_4^+	1.05 (0.84-1.32)	.66	1.32 (0.98-1.79)	.07	0.09 (0.06-0.15)	<.001[b]	5.53 (1.21-25.34)	.03[b]
BC	3.09 (2.28-4.20)	<.001[b]	1.09 (0.81-1.47)	.57	1.00 (0.81-1.24)	.97	1.96 (1.56-2.47)	<.001[b]
OM	1.28 (1.18-1.39)	<.001[b]	1.03 (0.97-1.10)	.33	0.96 (0.93-0.99)	.02[b]	0.98 (0.92-1.06)	.66
SS	4.53 (3.05-6.72)	<.001[b]	1.03 (0.82-1.30)	.80	1.24 (0.78-1.97)	.37	1.00 (0.57-1.75)	.99
Soil	0.90 (0.60-1.36)	.62	0.95 (0.71-1.27)	.74	0.92 (0.70-1.22)	.56	1.21 (0.94-1.55)	.14
MC	1.19 (1.03-1.38)	.02[b]	0.88 (0.77-1.01)	.08	0.76 (0.70-0.84)	<.001[b]	1.01 (0.94-1.09)	.76

Abbreviations: BC, black carbon; CARE-PF, Canadian Registry for Pulmonary Fibrosis; HR, hazard ratio; MC, multiconstituent; NH_4^+, ammonium; NO_3^-, nitrate; OM, organic matter; PFF, Pulmonary Fibrosis Foundation; $PM_{2.5}$, particulate matter 2.5 μm or less in diameter; Simmons, Simmons Center for Interstitial Lung Disease Registry; SO_4^{2-}, sulfate; SS, sea salt.

[a] Death and transplant were considered composite outcomes. Mortality or transplant effect size estimates for 5-year time-varying exposures to $PM_{2.5}$ calculated for each patient from enrollment to censoring. Results of complete models adjusting for enrollment year, age at enrollment, sex, race, smoking history, neighborhood disadvantage score, and site (in PFF and CARE-PF cohorts, which both comprised multiple sites) are included. One-stage meta-analysis also includes clustering by cohort and a disadvantage variable that was normalized across the 3 cohorts. Hazard ratios are reported per 1-μg/m^3 increase in $PM_{2.5}$ or constituents, except for MC models, where HRs reflect effect estimates per 1-quantile increase in the $PM_{2.5}$ mixture of all constituents.

[b] Significant associations.

PubMed®

tiny particles big health impacts

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> Am J Respir Crit Care Med. 2024 Aug 30. doi: 10.1164/rccm.202407-1476ED. Online ahead of print.

Tiny Particles, Big Health Impacts

John R Balmes [1 2], Nadia N Hansel [3]

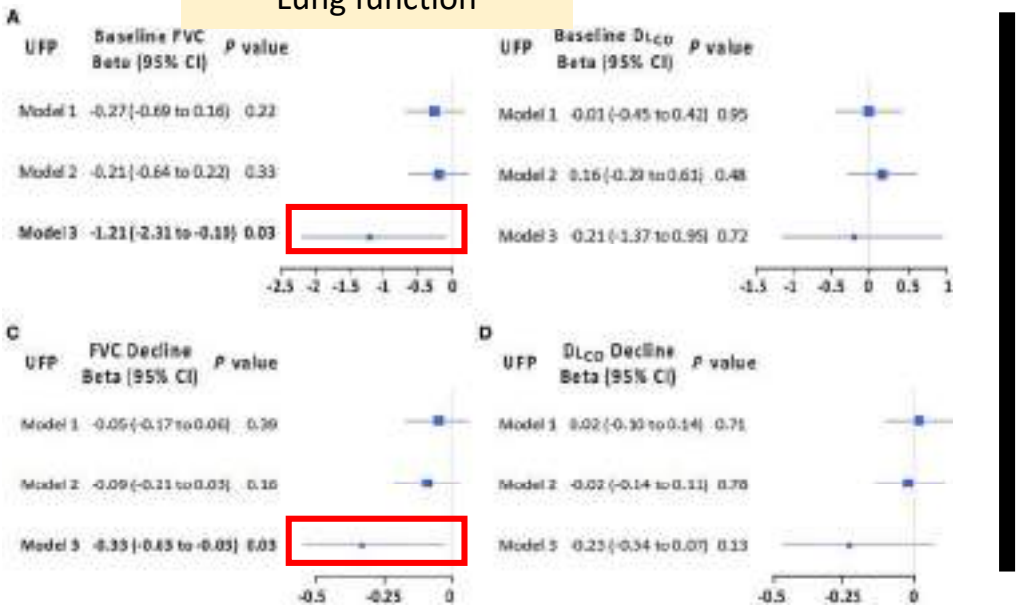
UltraFineParticles and IPF

Ambient Ultrafine Particulate Matter and Clinical Outcomes in Fibrotic Interstitial Lung Disease

Gillian C. Goobie[1,2,3,5], Provat K. Saha[6,7,8], Christopher Carlsten[1,2,3,5], Kevin F. Gibson[4,5], Kerri A. Johannson[10], Daniel J. Kass[6,7], Christopher J. Ryerson[1,3,9], Yingze Zhang[6,7], Allen L. Robinson[6,7], Albert A. Presto[6,7], and S. Mehdi Nouraie[6,8]

Increased UFP exposure associated with baseline FVC and transplantation-free survival

Lung function



Transplantation-Free Survival

Continuous UFP Model	Residential Address Exposure HR	95% CI	P	n
Model 1	1.01	0.99–1.04	0.31	1,416
Model 2	1.01	0.98–1.03	0.57	1,372
Model 3	**1.08**	**1.01–1.15**	**0.02**	1,372

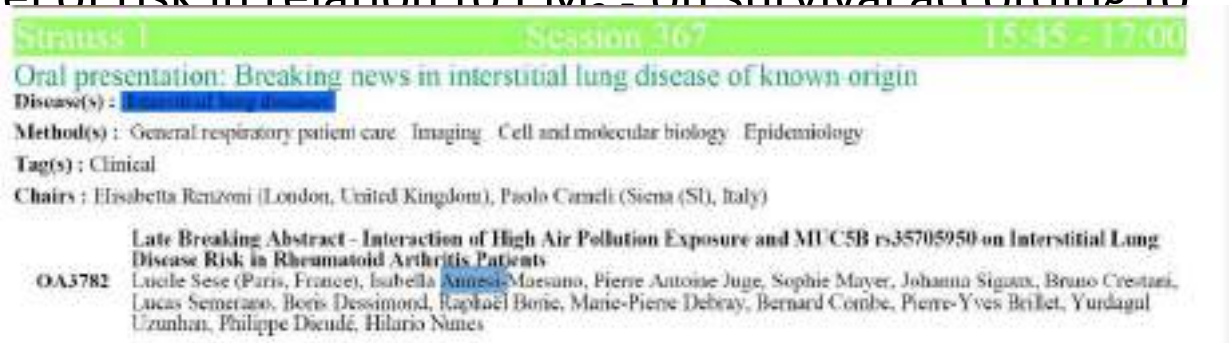
Associations of each 1,000 particles/cm3 increase in UFP matter exposure using the residential address linkage approach. Mmodel 1 (unadjusted), model 2 (adjusted for sex, age at enrollment, race, smoking history, and a socioeconomic covariate), and model 3 (model 2 with additional adjustment for continuous exposures to $PM_{2.5}$ µm and NO_2 by the same model)

Goobie et al. Am J Respir Crit Care Med, 2024

Air pollution, genetic susceptibility and IPF

- 3-fold risk of developing IPF in individuals with high genetic susceptibility to IPF (including *MUC5B* promoter polymorphism) and living in areas of heavy air pollution compared with those with low genetic susceptibility and low air pollution exposure (Cui, ERJ 2022)

- No increased level of risk in relation to $PM_{2.5}$ on survival according to *MUC5B* genotyp


Strauss 1 Session 367 15:45 - 17:00

Oral presentation: Breaking news in interstitial lung disease of known origin

Disease(s) : [redacted]

Method(s) : General respiratory patient care Imaging Cell and molecular biology Epidemiology

Tag(s) : Clinical

Chairs : Elisabetta Renzoni (London, United Kingdom), Paolo Cameli (Siena (SI), Italy)

OA3782 **Late Breaking Abstract - Interaction of High Air Pollution Exposure and MUC5B rs35705950 on Interstitial Lung Disease Risk in Rheumatoid Arthritis Patients**

Lucile Sese (Paris, France), Isabella Annesi-Maesano, Pierre Antoine Juge, Sophie Mayer, Johanna Sigaux, Bruno Crestani, Lucas Semerano, Boris Dessimond, Raphaël Borie, Marie-Pierre Debray, Bernard Combe, Pierre-Yves Brillet, Yurdagul Uzunhan, Philippe Dieudé, Hilario Nunes

Pathogenesis of IPF

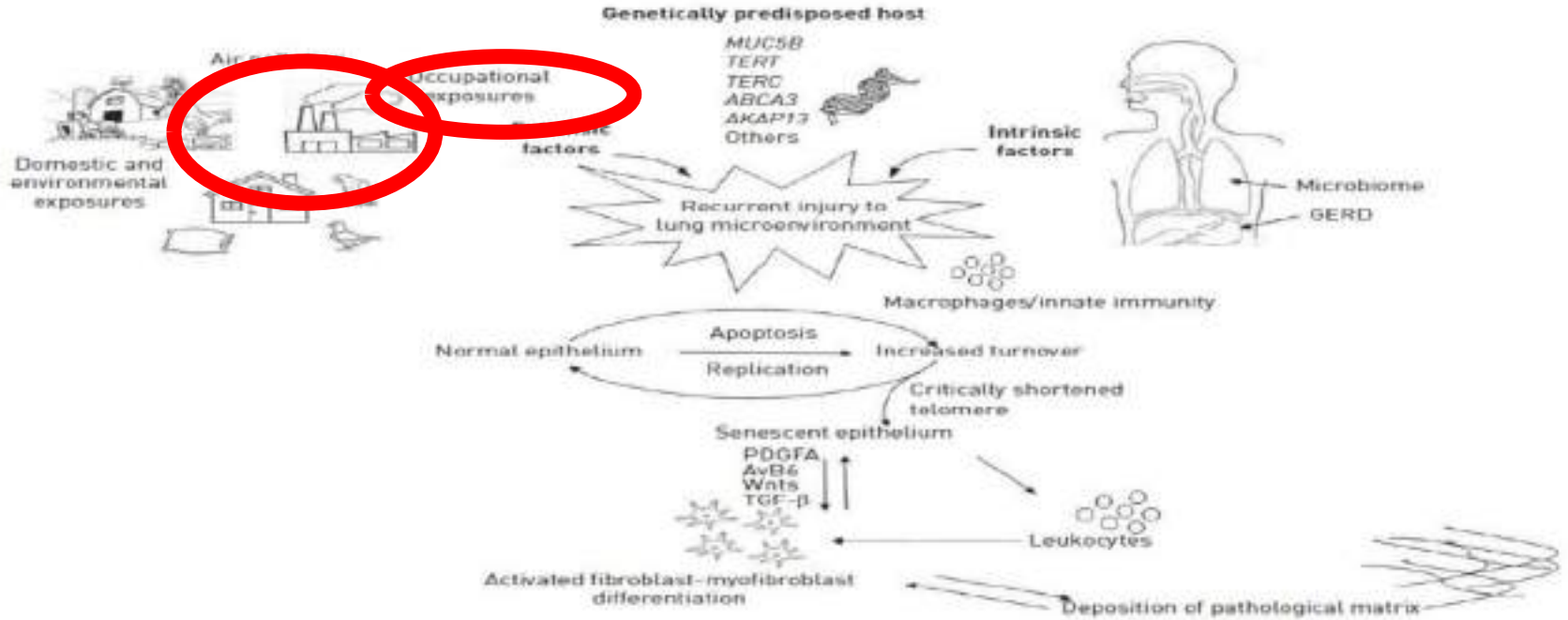
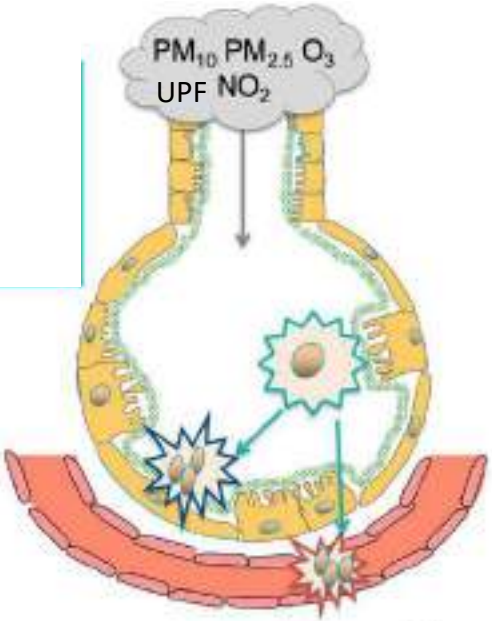


FIGURE 1 Proposed model for the pathogenesis of idiopathic pulmonary fibrosis. Extrinsic and intrinsic environmental exposures to the lung microenvironment cause recurrent airway injury. In the genetically predisposed host (mutations in mucin 5B (*MUC5B*), telomerase reverse transcriptase (*TERT*), telomerase RNA component (*TERC*), ATP binding cassette sub-family A3 (*ABCA3*), A-kinase anchor protein 13 (*AKAP13*), etc.), this cycle of increased epithelial turnover eventually leads to reprogramming to senescent epithelium. Fibroblasts are activated via pro-fibrotic mediators (platelet-derived growth factor subunit A (PDGFA), activating integrin B6 (avB6), Wnts, transforming growth factor-β (TGF-β)) that are released directly from abnormal epithelium, the activated innate immune system and leukocytes. Once activated, fibroblasts deposit pathological matrix, which leads to myofibroblast differentiation and progressive fibrosis. GERD: gastro-oesophageal reflux disease. Reproduced from [4] with permission.

- Sack C, Raghu G. Idiopathic pulmonary fibrosis: unmasking cryptogenic environmental factors. Eur Respir J. 2019 Feb 21;53(2):1801699. doi: 10.1183/13993003.01699-2018. PMID: 30487201.

Air Pollution mechanisms in IPF

Genetic susceptibility



Johannson et al. *Chest* 2015

Air pollutants exposure

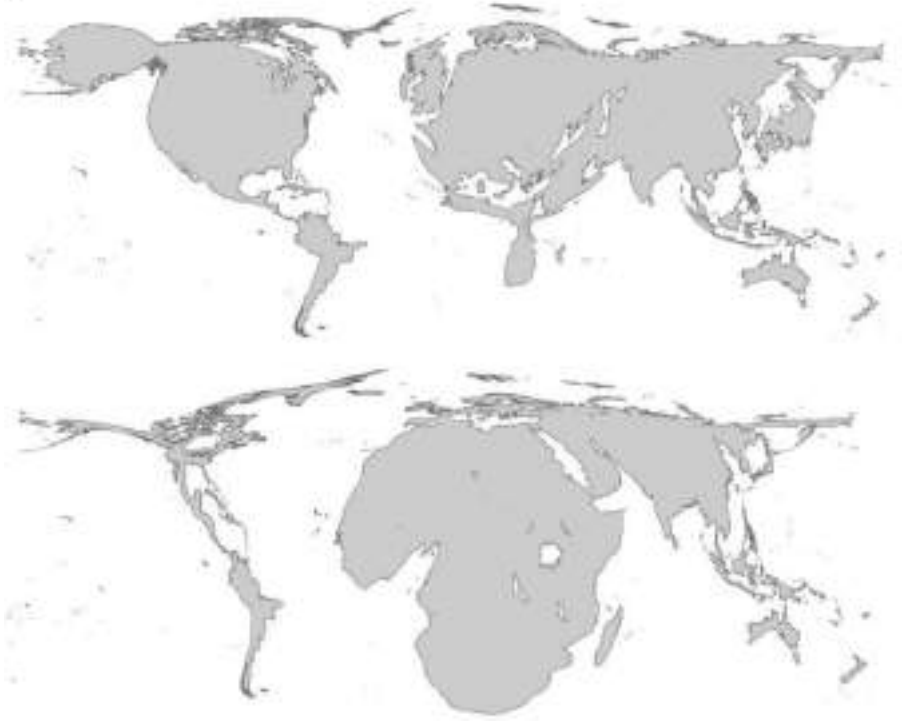
Oxidative stress
Mitochondrial dysfunction
Telomere shortening
Cellular senescence
Extracellular matrix remodeling

Disease development
Disease severity
Accelerated progression
Increased hospitalization risk
Increased acute exacerbation risk
Increased mortality

SOCIAL AND ENVIRONMENTAL INEQUITIES

HEALTH IMPACTS ARE UNFAIRLY DISTRIBUTED

Sharp increase in greenhouse gas (GHG) emissions began with the **Industrial Revolution** in the late 18th and early 19th centuries.



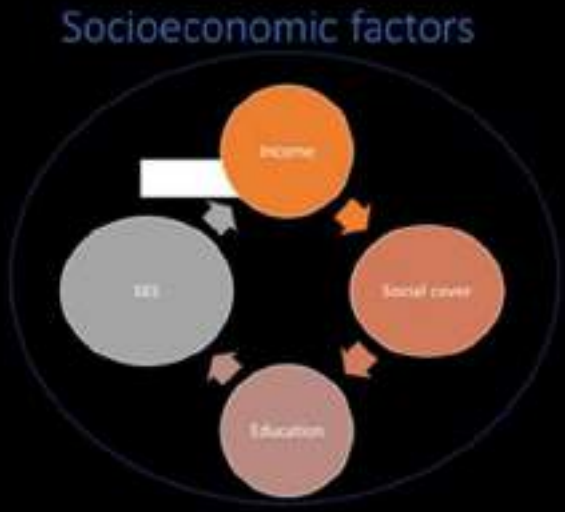
Cumulative emissions of greenhouse gases

WHO estimates of *per capita* mortality from climate change

Map projections from
Patz et al, 2007; WHO, 2009.

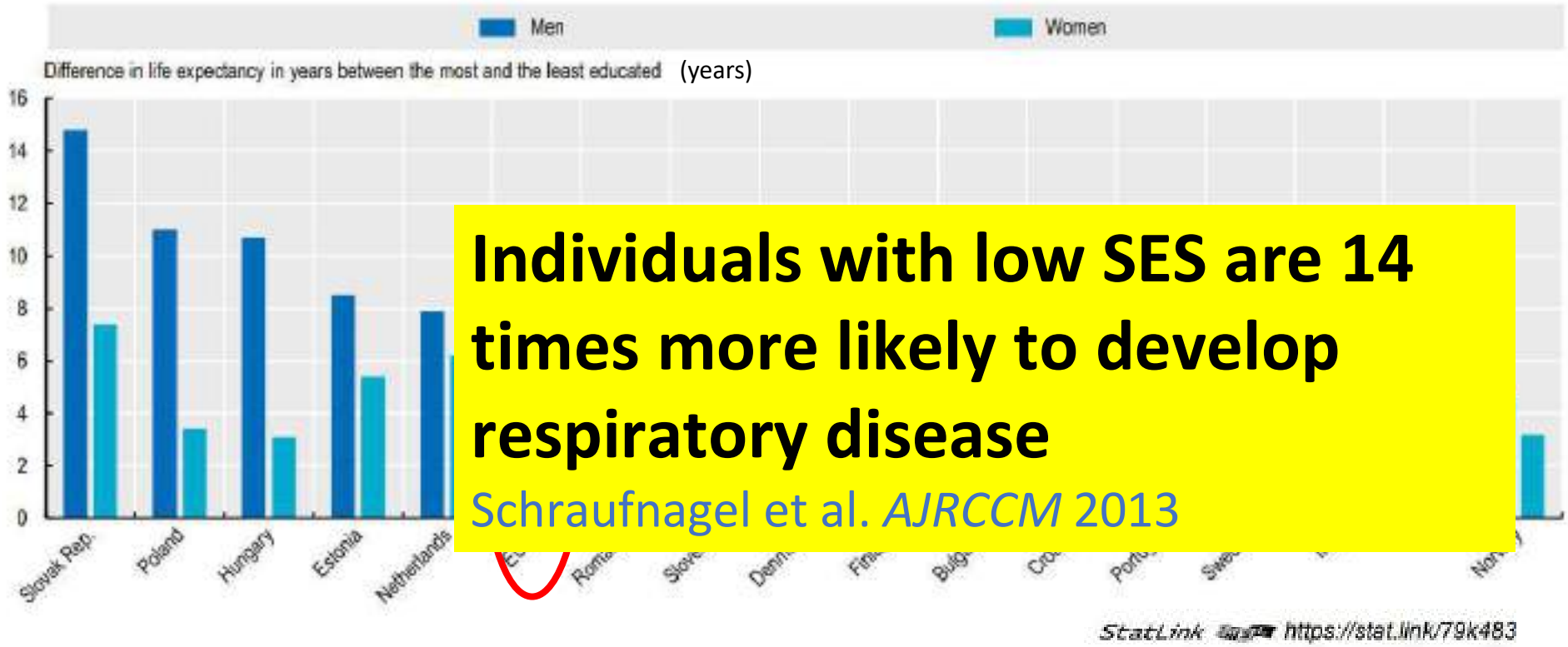
How does climate change affect health?

→ Environmental injustice!



IPF AND POVERTY

Gap in life expectancy between people with the highest and lowest level of education in EU 2017, by gender



Individuals with low SES are 14 times more likely to develop respiratory disease

Schraufnagel et al. *AJRCCM* 2013

SES and IPF outcomes

- ❑ Few studies

IPF Patients with low income have reduced health care cover and less transplantation but a similar mortality.

Table 2 Adjusted odds of four outcomes in IPF patients (ICD9 516.3 or 515)[a]

	Lung Transplant		Death		Rehabilitation Transfer		VATS Biopsy	
	Odds Ratio (95% CI)	P-Value	Odds Ratio (95% CI)	P-Value	Odds Ratio (95% CI)	P-Value	Odds Ratio (95% CI)	P-Value
Insurance								
Non-medicaid	*Reference*		*Reference*		*Reference*		*Reference*	
Medicaid	0.30 (0.16, 0.57)	< 0.001	1.00 (0.89, 1.12)	0.95	0.53 (0.33, 0.85)	0.01	0.46 (0.35, 0.60)	< 0.001
Uninsured	0.22 (0.07, 0.72)	0.01	1.12 (0.92, 1.35)	0.26	0.41 (0.18, 0.93)	0.03	0.52 (0.35, 0.77)	< 0.01
ZIP Income Quartile								
Quartile 1	0.46 (0.32, 0.66)	< 0.001	0.93 (0.87, 1.00)	0.04	1.16 (0.92, 1.45)	0.21	1.01 (0.81, 1.25)	0.96
Quartile 2	0.56 (0.43, 0.73)	< 0.001	0.91 (0.85, 0.97)	< 0.01	1.00 (0.80, 1.24)	0.97	1.09 (0.90, 1.33)	0.38
Quartile 3	0.71 (0.57, 0.89)	< 0.01	0.96 (0.90, 1.02)	0.19	1.11 (0.90, 1.36)	0.33	0.98 (0.81, 1.18)	0.81
Quartile 4 (Highest)	*Reference*		*Reference*		*Reference*		*Reference*	

Gaffney et al. *BMC Health Services Research* 2018

SES and IPF outcomes

> Am J Respir Crit Care Med. 2021 Nov 24. doi: 10.1164/rccm.202109-2065OC.
Online ahead of print.

Neighborhood-level Disadvantage Impacts on Patients with Fibrotic Interstitial Lung Disease

Gillian C Goobie [1, 2], Christopher J Ryerson [3], Kerri A Johannson [4], Erin Schikowski [5], Richard H Zou [6], Nasreen Khalil [3], Veronica Marcoux [7], Deborah Assayag [8], Hélène Manganas [9], Jolene H Fisher [10], Martin Rj Kolb [11], Kevin F Gibson [12], Daniel J Kass [13], Yingze Zhang [14], Kathleen O Lindell [15], S Mehdi Nouraie [13]

4729 patients with fibrosing ILD in Canadian and USA registries with identified deprivation index

- Greater disadvantage associated with reduced baseline DL_{CO}
- In USA only: fILD with the greatest disadvantage (top quartile) experience the highest risk of mortality (HR =1.51, p=0.002)
  - IPF : disadvantaged related to less transplantation (OR =0.46, p=0.04)

SES Deprivation and telomere length

Contents lists available at ScienceDirect

SSM - Population Health

journal homepage: http://www.elsevier.com/locate/ssmph

Article

The relationship between neighborhood socioeconomic deprivation and telomere length: The 1999–2002 National Health and Nutrition Examination Survey

Tiffany M. Powell-Wiley, Samson Y. Gebreab, Sophie E. Claudel, Colby Ayers, Marcus R. Andrews, Joel Adu-Brimpong, David Berrigan, Sharon K. Davis

5 106 IPF patients

- Precariousness inversely correlated with telomere length

→ Precariousness can contribute to telomere length (stress, unhealthy lifestyles, etc.)

IPF, AIR POLLUTION AND POVERTY

Air pollution and SES

Curr Envir Health Rpt (2015) 2:440–450
DOI 10.1007/s40572-015-0069-5

AIR POLLUTION AND HEALTH (JD KAUFMAN AND SD ADAR, SECTION EDITORS)

Socioeconomic Disparities and Air Pollution Exposure: a Global Review

Anjum Hajat[1] · Charlene Hsia[2] · Marie S. O'Neill[3]

- Socially disadvantaged groups are likely to live in areas with higher levels of air pollution.
- Precariousness can potentiate the deleterious effect of exposure

Original research

Relative environmental and social disadvantage in patients with idiopathic pulmonary fibrosis

Na'ama Avitzur,[1] Elizabeth M Noth,[2] Mubasiru Lamidi,[3] Steven D Nathan,[4] Harold R Collard,[5] Alison M DeDent,[5] Neeta Thakur,[5] Kerri A Johannson [1,6]

Thorax: first published as 1

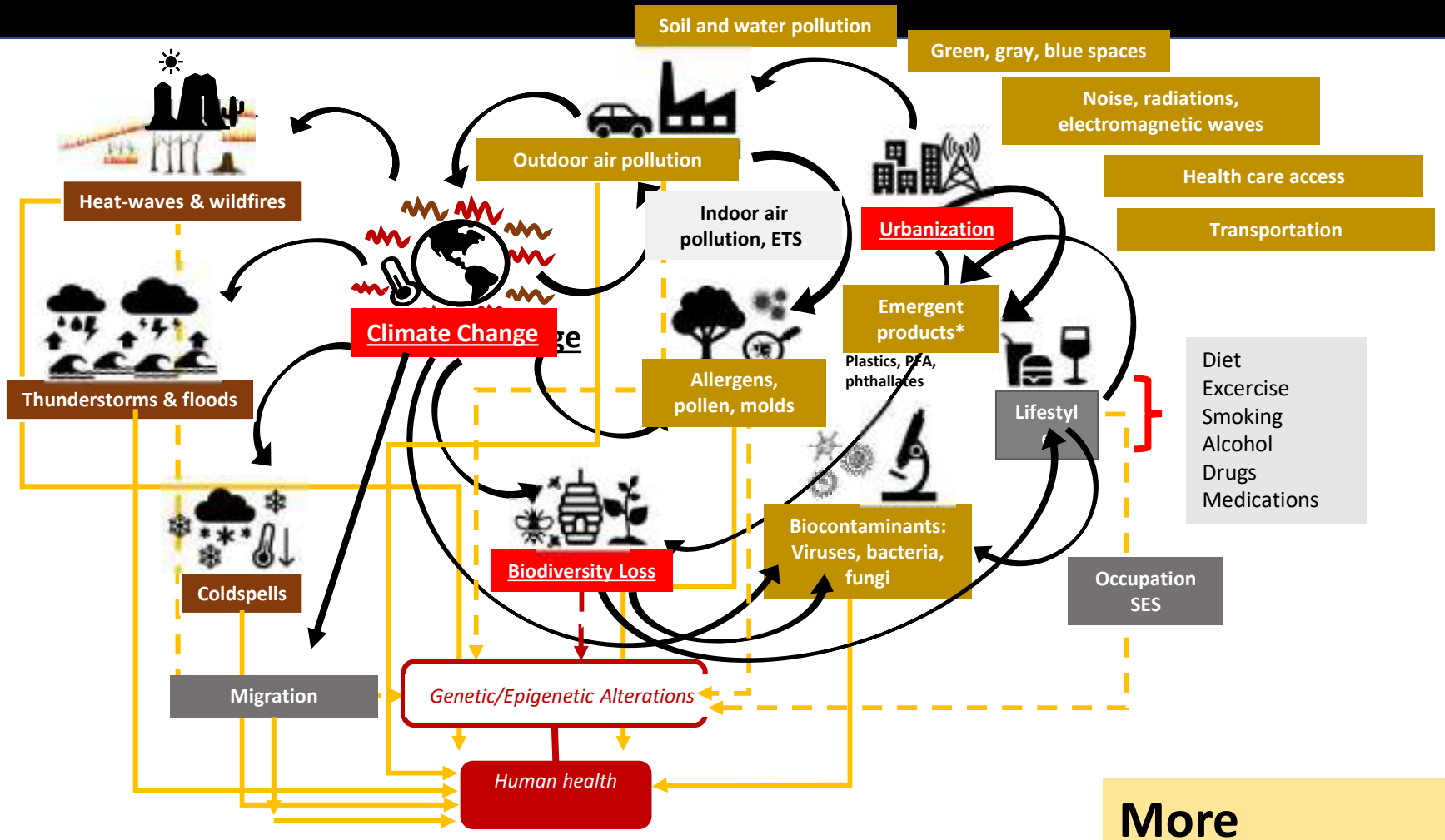
CalEnviroScreen 3.0 (CES) quantifying environmental burden combining population, environmental and pollution vulnerability

In IPF patients:

- Higher environmental exposures and vulnerability (CES) associated with:
  - lower baseline lung function and lower antifibrotic use
  - mortality (but not in sensitivity analyses)

IPF, AIR POLLUTION, POVERTY, AND MUCH MORE

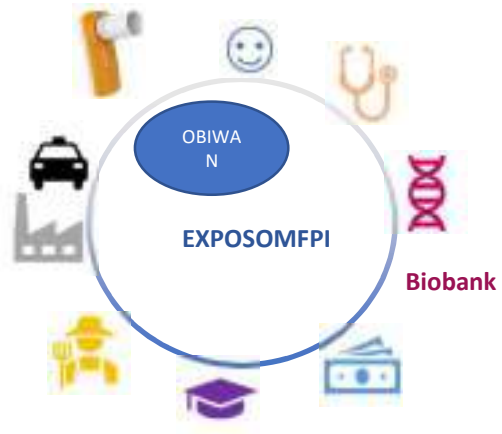
Approaching the exposome to better understand fILD



ETS: Environmental Tobacco Smoking; *: plastic, PFAS, etc.

More susceptible/ vulnerable individuals

EXPOSOMFPI



Investigateur Principal : Dr Sesé
Responsable scientifique: Pr Nunes
Cohorte nationale multicentrique
FPI : n = 200 (avril 2021)
Suivi prospectif 2 ans

Equipe 1 : Recherche clinique
Pr Nunes, Pr Planès, Dr Gille, Pr Levy, Pr Ziol, Pr Cottin, (URC, CRB, CRMR Orphalung, INSERM U-1272)

Equipe 2 : Pollution de l'air / Exposome
Pr Annesi Maesano (INSERM UA-1318, IDESP) Boris Dessimond

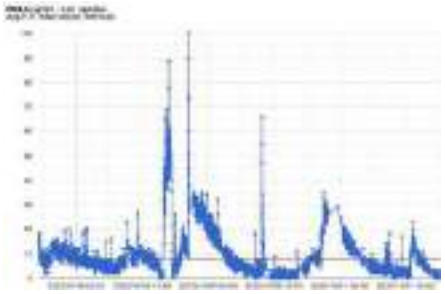
Equipe 3 : Expositions professionnelles
Pr Paris, Pr Jouneau, Pathologies professionnelles et environnementales (INSERM U-1085)

Equipe 4 : Sociologie
Pr Rosental, Dr Cavalin (CNRS, Sciences Po Paris)

OBIWAN-FPI: OBjects In Wide Area Network for IPF

Etude de l'impact de la pollution individuelle (intérieure et extérieure) sur l'évolution de la fonction respiratoire dans la FPI

Objets connectés



Investigateur Principal : Dr Sesé

- Etude pilote monocentrique
- FPI : n = 30
- Suivi prospectif : 1 an

Partenaires :

Pr Nunes, Pr Annesi Maesano, Dr Gille, Pr Planès, M. Dessimond

Association de patients : **APEFPI**

Established and postulated impacts of climate change in patients with interstitial lung disease (ILD)

- Very likely through air pollution, temperature and social factors

- Paper submitted to Lancet Respiratory Medicine

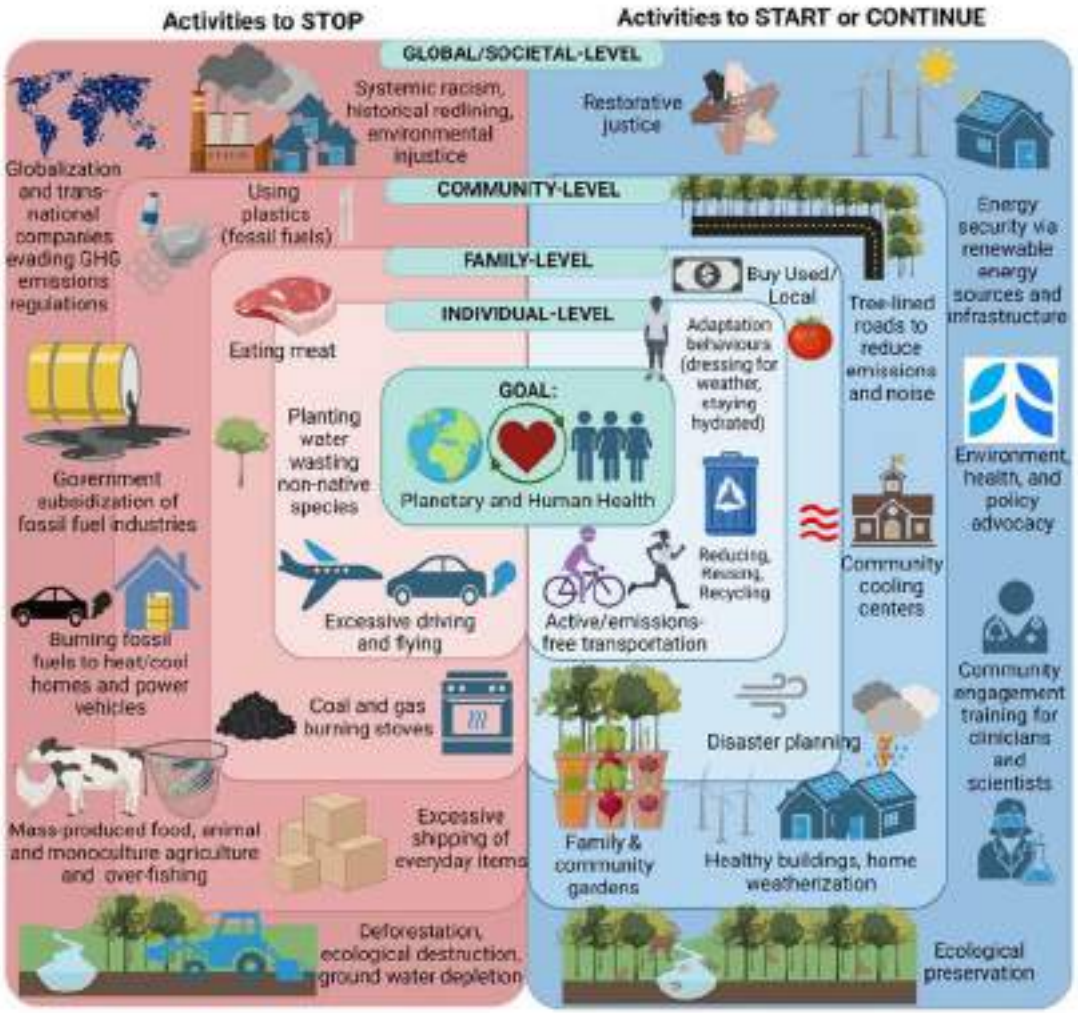
Implications for ipf Patient Care

Understanding the link between IPF and the exposome is crucial for developing effective treatment and prevention strategies. Efforts should focus on:

- **Early detection and intervention:** Implementing screening programs in underserved communities.
- **Improving access to care:** Expanding healthcare coverage and reducing financial barriers to treatment.
- **Addressing environmental factors:** Promoting policies to reduce air pollution and occupational hazards in low-income communities.
- **Addressing environmental injustices**
- **Patient support programs:** Providing financial assistance and support services for patients with IPF.

CLIMATE CHANGE INCREASES THE BURDEN

Activities to Stop (left, red background) and Activities to Start (right, blue background) to Address EJ in the Climate Change era




AMERICAN THORACIC SOCIETY DOCUMENTS

Climate Change and Respiratory Health: Opportunities to Contribute to Environmental Justice

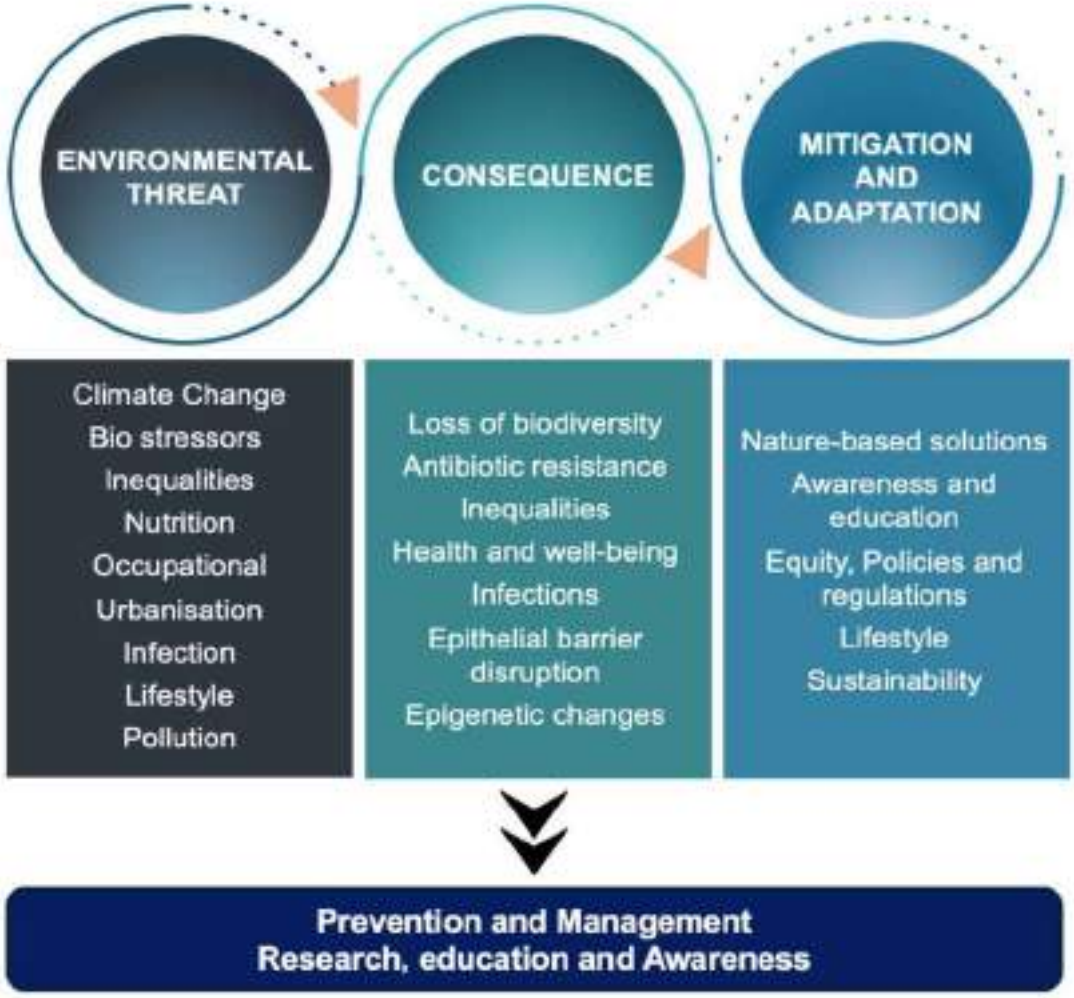
An Official American Thoracic Society Workshop Report

Vulnerable individuals are more exposed to environmental hazards

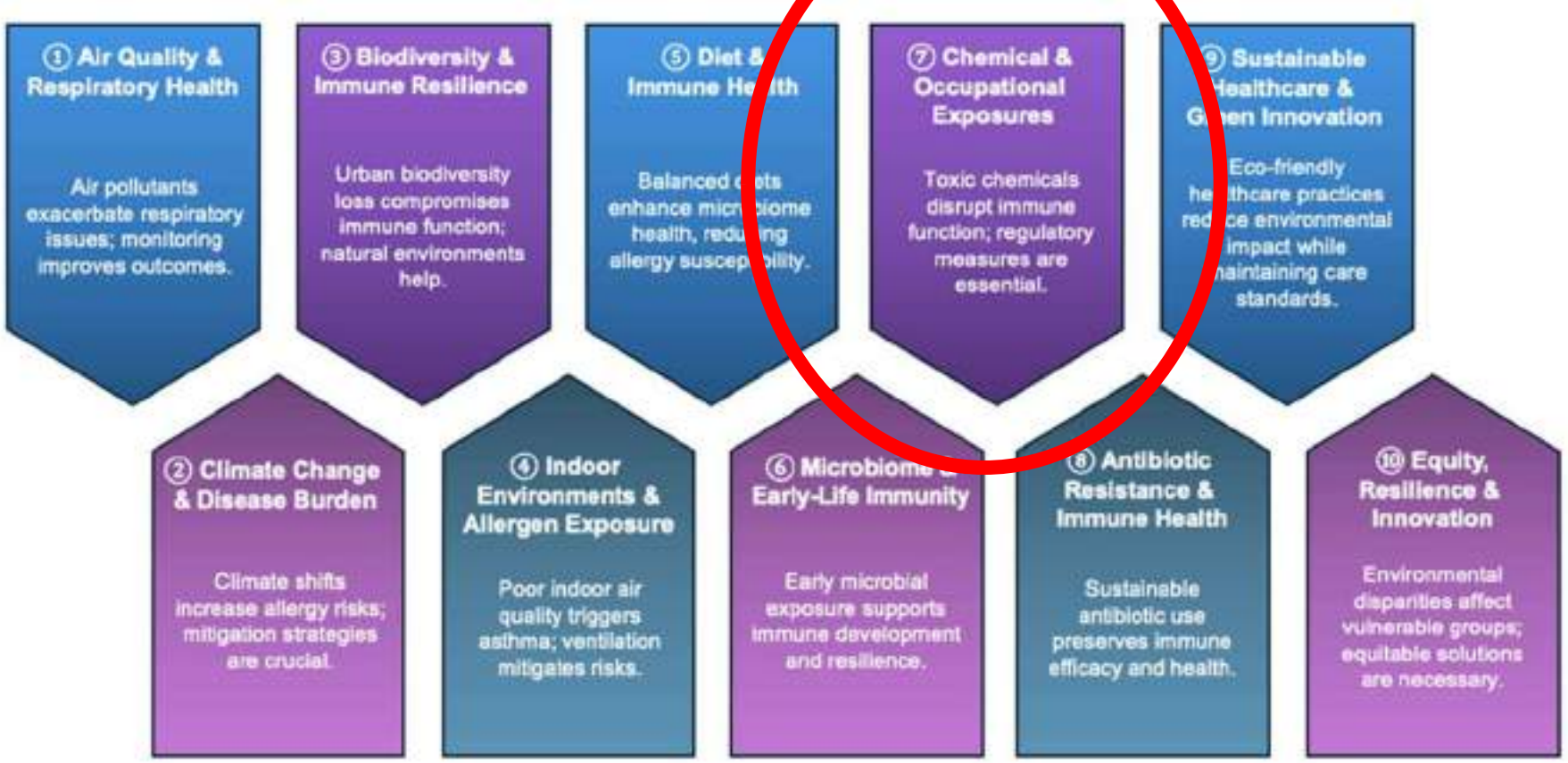
Annals ATS 2025

Take home messages

- Rising temperatures, air pollution, extreme weather events, and shifting disease patterns can increase respiratory diseases, heat stress, and occupational hazards, disproportionately affecting vulnerable populations.
- Understanding climate-driven health risks in communities and workplaces is crucial for protecting public health, ensuring worker safety, and developing effective adaptation strategies.
- Proactive measures, including policy interventions, workplace adaptations, and community resilience planning, are essential to mitigate these risks and safeguard long-term health and productivity.


ENVIRONMENTAL THREAT
CONSEQUENCE
MITIGATION AND ADAPTATION
Climate Change
Bio stressors
Inequalities
Nutrition
Occupational
Urbanisation
Infection
Lifestyle
Pollution
Loss of biodiversity
Antibiotic resistance
Inequalities
Health and well-being
Infections
Epithelial barrier disruption
Epigenetic changes
Nature-based solutions
Awareness and education
Equity, Policies and regulations
Lifestyle
Sustainability
Prevention and Management
Research, education and Awareness

EAACI
EUROPEAN ACADEMY OF ALLERGY AND CLINICAL IMMUNOLOGY



Immun4Cure

N=40

Climate change and respiratory disease: European Respiratory Society position statement

Climate change and respiratory diseases

REVIEW

Thunderstorm-related asthma: what happens and why

Projections of the effects of climate change on allergic asthma: the contribution of aerobiology

Allergy, 2010

L. Cecchi[1,*], G. D'Amato[2,*], J. G. Ayres[3,*], C. Galan[4], F. Forastiere[5,*], B. Forsberg[6,*], J. Gerrits C. Nunes[8,*], H. Behrendt[9,*], C. Akdis[10,*], R. Dahl[11] & I. Annesi-Maesano[12,*]

Allergy EUROPEAN JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY

EDITORIAL Free Access

Climate change: A call to action for the United Nation

Karl C. Nadeau, Ioana Agache, Marek Jutel, Isabella Annesi Maesano, Nilgiocial Akdis, Vanitha Sampath, Gennaro D'Amato, Lorenzo Cecchi, Claudia Traidl-Hoffmann, Cezmi A.

First published: 03 September 2021 | https://doi.org/10.1111/all.15079 | Citations 17

Climate Change, Migration, and Allergic Respiratory Diseases: An Update for the Allergist

WAO journal, 2011

Gennaro D'Amato (Chair), MD, Menachem Rotem, ●●●, Ronald Dahl, ●●●, Michael Blaiss, ●●●, Ernesto Ridolo, ●●●, Lorenzo Cecchi, ●●●, Nelson Rosario, ●●●, Cassin Moulla, ●●●, Ignacio Antotegui, ●●●, Isabella Annesi-Maesano, ●●●, for the WAO Climate Change and Migration and Allergy Special Committee

WAO journal, 2011

REVIEW

Meteorological conditions, climate cha new emerging factors, and asthma an related allergic disorders. A statement the World Allergy Organization

JACI, 2018

Mechanisms of allergic diseases

External exposome and allergic respiratory and skin diseases

Journal of Allergy and Clinical Immunology

Volume 148, Issue 6, December 2021, Pages 1386-1377

Reviews and feature article

Climate change and global issues in allergy and immunology

Busen E. Pacheco MD *, Guillermo Guidos-Fogelbach MD, PhD, Isabella Annesi-Maesano MD, PhD, DSc, Ruby Pawankar MD, PhD

The effects of climate change on respiratory allergy and asthma induced by pollen and mold allergens

AMERICAN THORACIC SOCIETY DOCUMENTS

Climate Change and Respiratory Health: Opportunities to Contribute to Environmental Justice

An Official American Thoracic Society Workshop Report

To my PhD students
Since 2015
















isabella.annesi-maesano@inserm.fr